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BRITISH JOURNAL OF PREVENTIVE & SOCIAL MEDICINE

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THE SELF-CONTROLLED AND SELF-RECORDED CLINICAL TRIAL FOR LOW-GRADE MORBIDITY

BY

LANCELOT HOGBEN and M. SIM

Birmingham

1. Introduction

Medicine has hitherto advanced almost exclusively by study of:

(*a*) the individual patient in the consulting room, in the clinic, or in the ward;

(*b*) groups of comparable individuals subjected to different treatments or to different risks.

That we have learned much about controllable agencies contributory to the spread of disease by the group method has long been recognized. That reliance on the clinician's experience of the response of individuals to a particular treatment may lead to unjustifiable conclusions, if without due regard to the principles of *controlled* experimentation, is also indisputable; and it has lately become a widely accepted dogma that a truly scientific evaluation of the efficacy of therapeutic or prophylactic measures is possible only by recourse to:

(*a*) recorded responses of treatment and control groups of individuals selected randomwise with or without pairing with reference to putatively relevant variables;

(*b*) standard statistical tests to validate conclusions referable to comparisons based thereon.

Doubtless the adoption of this discipline has been a salutary check to rash announcements and has promoted a more punctilious regard for rules of evidence. Even so, the claims advanced for it are not beyond the reach of legitimate criticism. The now current recipe for a clinical trial based on group comparison sets out a balance sheet in which individual variability with respect both to nature and to previous nurture does not appear as an explicit item in the final statement of the account; but such variability of response to treatment may be of paramount interest in practice. Partly for this reason, it is difficult to confer any sharp definition on the hypothetical infinite population conceptualized as the source of samples of tested individuals in the theory of any current statistical procedure for validating a comparison. By the same token, it is difficult to specify the precise class of situations to which conclusions endorsed by such procedures are relevant. Nor is it immaterial to mention that the credentials of statistical tests most commonly invoked in this context have lately become the focus of a vigorous controversy which admits no prospect of easy compromise.

In the context of the clinical trial, the word *control* has all the emotive force of a tradition associated with the advance of physiology to the status of an exact science. This being so, it is fitting to re-examine the disposition to identify controlled experimentation exclusively with group comparison. A little reflection will suffice to discredit the assumption that inquiry limited to successive observations based on a single individual is indeed foreign to the pattern of a rigorously controlled experiment in the biological laboratory. Indeed, a single illustration of a technique which has greatly advanced our understanding of the ductless glands, the control of the circulation, and the mechanism of respiratory movements will make it abundantly clear that exclusive reliance on methods amenable to statistical treatment of groups is inconsistent with laboratory practice universally endorsed since Oliver and Schafer (1894–95) discovered the vasoconstrictor action of extracts of the posterior lobe of the hypophysis and of the adrenal medulla.

Over a wide field of research, the physiologist has long exploited the method of successive differences here called *self-controlled* experimentation, as in inquiries of the sort last mentioned. A now familiar pattern is as follows:

We prepare an extract from a particular tissue suspected as the seat of production or storage of an active component, we also make extracts (E_1, E_2, E_3, etc.) of a wide range of other tissues by exactly the same method. We successively introduce in exactly the same way equal quantities of T and of E_1, E_2, etc. into the circulation with equally spaced intervals of recovery in an order which excludes masking an authentic or disclosing

a spurious effect by reason either of delayed action or of an intrinsic rhythm. Finally, we record the appropriately defined response as + or −. We then say that we have established the seat of the putative active substance if some such record as follows is the outcome:

Treatment	E_1	T	E_2	T	E_1	T	E_3	E_4	T	T	E_5	T	E_2
Response	−	+	−	+	−	+	−	−	+	+	−	+	−
Treatment	E_1	E_6	T	E_5	T	T	T	E_3	T	E_6	T	T	E_4
Response	−	−	+	−	+	+	+	−	+	−	+	+	−

One-to-one correspondence of stimulus and response is here the criterion of a conclusive outcome, and statistical assessment in the current sense of the term is not relevant to its interpretation. What is essentially relevant is the precise specification of all the precautions requisite to correct interpretation, *e.g.* washing the last remnants from the syringe and attached vein into the circulation by immediate injection of an exactly equal quantity of saline after the injection of each extract. Needless to say, the word *exactly* in this context is shorthand for *as nearly as possible with punctilious regard for every conceivably relevant circumstance.* Thus we must concede that the background knowledge, character, and training of the investigator are themselves relevant to our confidence in the validity of the final interpretation; but this *caveat* has no particular bearing on the thesis that statistical methods are essential to validification of conclusions drawn from experiment. It is indeed a mischievous delusion that statistical recipes can remedy incompetent observation. What sufficiently commends the *self-controlled* experiment, so interpreted without the *nihil obstat et imprimatur* of the statistician, is that firmly established conclusions separately referable to individuals with observable peculiarities can provide more refined data for evaluating the role of nature and of previous nurture in the characteristic response to a treatment.

To affirm the possibility of self-controlled experimentation on human subjects is by no means the statement of a new notion. Indeed, one recalls many well-thumbed records of discoveries made by individual investigators who have played the guinea-pig role in their own researches; but there is a pardonable disposition to regard unrecognized motivations of the investigator at work on his own person as inimical to the proper precautions on which the method relies. This misgiving is wholly justifiable, if the recorded response is not amenable to independent observation, as was true in its time of the work of Head, Rivers, and others (1920) on recovery of sensibility during regeneration following nerve section. On the other hand, self-experiment by no means circumscribes the conduct of self-controlled experimentation. This we realize at once, when we recall now sufficiently familiar war-time studies on inanition carried out with the co-operation of Quaker volunteers. What justifies the foregoing excursus is that there is still a disposition to regard the method itself with suspicion, to undervalue its merits, to exaggerate the obstacles to employing it or to understate the range of situations in which no such obstacles exist.

If we approach the problem against a background in which the major advances of medicine occurred in the social setting of pestilences, practical difficulties of enlisting volunteers without violence to ethical standards are easy to exaggerate; but a wholesome regard for the sanctity of life becomes increasingly less relevant to a statement of the priorities of medicine in western communities with a life-expectation well above 60 years at birth. There is indeed an ever more pressing need for knowledge about many once-thought *minor* evils. We cannot effectively tackle them by group methods; but we might well do so with the help of individuals now willing to risk life or limb on the speedway or in the ascent of Mount Everest, if we could institutionalize a not uncommon appetite for adventure consistent with acceptable moral standards. It is not beyond the reach of social engineering to create a new corps of civil defence with that end in view. The voluntary response to the appeal for blood donors disposes of doubts about the feasibility of such an undertaking, if sponsored by an adequately accredited organization.

Doubts concerning the reliability of evidence obtained by the method of self-controlled experimentation on human subjects call for serious consideration in so far as:

(*a*) we may need to rely on the testimony of the subject for the recording of the relevant response;

(*b*) both the response *per se* and the reliability of the record may be influenced by stimuli which operate at a *cognitive* and/or *emotive* level.

Each of these considerations calls for directives more explicit than the proper conduct of animal experiment demands. To forestall misinterpretation arising from the subject's knowledge of the aim and the subject's recognition of the pattern of the trial, it will not suffice to keep the subject in ignorance of the order of presentation of stimuli. In a prolonged trial—and such trials will commonly continue over a long period—any regularity of the pattern may provide clues to recognition. Thus the order of presentation must be intrinsically *chaotic;* and the

method of presentation must provide the subject with no lead to the identification of the stimulus. Concerning the order, there seems to be no particular reason to adopt the device of attaching random numbers to the stimuli (*e.g.* tablets), as we should deem to be desirable in the proper domain of stochastic inference. On the contrary, intentional disorder, *i.e.* a pattern assigned to make easier the recognition of delayed action and to forestall misinterpretation of a normal rhythm would appear to be a more reliable way of achieving the end in view.

As regards concealment of more explicit clues, the following procedure is commendable:

(*i*) the *physician-investigator* supervises the allocation of code numbers or letters to the stimuli for presentation in the pre-assigned order;

(*ii*) an *intermediary* (*e.g.* nurse, orderly, houseman) equipped with the code signs and the programme of presentation has no knowledge of the code;

(*iii*) the *subject* who receives the stimulus from the intermediary does so without access to the code sign.

These indications do not suffice to dispose of all spurious regularities emergent at the level of cognition. It is pertinent to cite them as prior *desiderata* because realizable with little inconvenience, if recognized at the outset as such. If surmountable, the special difficulties which beset reliance on the subject's testimony to the appropriate content of the record and the subject's reliable rendition of same also brings into focus the too-little recognized merit of self-controlled experimentation in a field as yet little influenced by the austere regimen of the statistical method. We shall recognize it more clearly, if we are able to sidestep a misgiving which the physician may entertain, if unduly subservient to laboratory tradition. The laboratory worker is suspicious of reliance on verbal testimony, and rightly so if we fail to distinguish between statements of fact accessible to independent observation and statements of feeling not susceptible to independent inquiry. If we reject all verbal testimony for this reason, we shall impose a heavy burden on the physician-investigator. Accordingly, the time-consumption involved in keeping any single subject under observation for a long period will substantially compromise the claims of the method.

The procedure cited above as a type specimen of self-controlled experimentation on laboratory animals is *self-recorded* in so far as the kymographic signal set-up can incorporate all relevant observations. In so far as it is practicable to prescribe a regimen to ensure reliable and adequate data from a suitably designed document completed by the human subject, we may speak of it as *self-recorded and self-controlled* experimentation. If such a prescription is indeed practicable, it will be possible to undertake a comprehensive programme of research in hitherto neglected fields of low-grade morbidity by enlisting volunteer subjects in a *normal environment* without recourse to expenditure on living accommodation. In what follows we shall accordingly assume that:

(*i*) constant observation of the subject by the investigator is impracticable in the conduct of many worthwhile inquiries;

(*ii*) the patient's verbal responses constitute a worthwhile source of relevant information if rightly recorded and rightly interpreted;

(*iii*) our knowledge of referred pain suffices to make real the distinction between right recording and right interpreting without enlisting the reader as a partisan of an explicitly behaviourist outlook.

To get the highest measure of useful and usable co-operation from the subject at the level of reliable recording is the less interesting but not less essential part of our task. For this purpose, it will be necessary to design a daily record sheet:

(*a*) explicitly specifying *all* information required to be supplied by subject;

(*b*) explicitly indicating the time of day at which each item of information is required to be completed;

(*c*) set out in a form to ensure minimum effort in subsequent analysis by the investigator;

(*d*) expressed in terms carefully chosen to forestall any error in interpreting the intention with due regard to the intelligence and educational level of the subject.

(*e*) providing both internal and external means of checking the intrinsic value of the information recorded.

Needless to say, the choice of subjects will determine the scope of the testimony appropriately incorporated in the record. To the extent that it is possible to design records in conformity with these requirements, and more especially with the last, we shall open a new door to psychiatric investigation conducted in terms consistent with the more exacting standards of laboratory experiment on animals. The need to distinguish between internal and external checks on verbal testimony arises from two different levels at which it may be useful, and emphasizes the need for a more refined distinction than the traditional dichotomy of signs and symptoms. If the subject's testimony relates to events

which any other person would describe in the same terms, the record of his or her own verbal responses is directly assessable by external checks, and the mere fact that it is a statement about himself or herself (*e.g.* frequency of micturition) does not set it in a category apart from other observations. Another class of assertions relative to the *self* are not self-made observations in the commonly accepted sense of the term, *i.e.* in so far as the meaningful content is manifest at its face value; but the investigation of their consistent relation to the subject's past experience, bodily states, current environment in general, or treatment in particular, may lead to an interpretation which confers on them a hitherto unsuspectedly meaningful content, as the study of referred pain testifies.

For lack of a current term, we may speak of a verbal response of the second sort as *non-observational*. Though the interpretation may have little or no connexion with the *manifest form of words*, any regular association between external occurrences or observable bodily states and a verbal response at the non-observational level so defined may well be in its own right an intrinsically valuable addition to our knowledge of human behaviour. Regardless of its ostensible bearing on the end in view, it is therefore desirable that the record should incorporate as much observational testimony amenable to direct observation, and hence to external checking, as is consistent with limitations imposed by the educational level of the subject. This is indeed desirable both because it will provide additional data for validifying the reliability of the record, and because it will offer a background for comparison of non-observational verbal responses and overt behaviour of the sort loosely referred to as discharge of normal bodily functions. It is also desirable, but not always, for another reason. In some inquiries it may suffice for the end in view, if the identification of the treatment procedure does not emerge at the level of cognition. In others it may also be essential to keep the subject in ignorance of the intention, and it will be less easy to accomplish this, if the items of the proforma are exclusively relevant thereto.

Among data for the background picture of the subject's discharge of normal bodily functions and assessment of the subject's reliability as a clerical assistant in the investigation, times of defecation and micturition, body weight, times of retiring and rising, hours of work and physical exertions, are appropriate items of the self-record for subjects of any educational level. After brief preliminary tuition, there is no reason why intelligent volunteers with a good high school record should not also undertake to complete at suitably prescribed intervals entries for pulse and respiration rates, body temperature, and blood pressure.

If we are able to codify a satisfactory regimen of self-recorded testimony for self-recorded and self-controlled experimentation so conceived, we have also the means of interpreting regularities of non-observational verbal behaviour with vastly greater economy of effort than the task would otherwise demand. Where it is possible to enlist groups of two or more individuals in cohabitation, we can accommodate synchronization of the record of A's verbal response to his condition with B's verbal description of A's behaviour and *vice versa*. In this way it may be possible to establish a one-to-one correspondence between verbal response at the non-observational level and verbal response at the observational level. In so far as this is indeed possible, it will place at the disposal of the psychiatrist a dictionary of fact and feeling prerequisite to the installation of psychiatry as a branch of experimental medicine.

2. The Subject

In what follows, it is not our intention to exhibit the outcome of a self-controlled and self-recorded trial as a contribution to therapeutics. The intention is methodological. Our main concern is to exhibit the defects of an experiment undertaken without due consideration to design as a basis for prescribing a better procedure. None the less, a few preliminary remarks on the condition of the subject himself are pertinent.

Over a period of about 15 years, the subject had experienced thyroid disorder. Ten years before the experiment began he had undergone partial thyroidectomy for an extensive retrosternal goitre, and nine years later had a second operation for removal of a small median growth of thyroid tissue near the normal site of the isthmus. He had been complaining of increasing lassitude resulting in reduction of his customary activity; but physical investigations provided no grounds for attributing this to previous surgical treatment. His previous retrosternal goitre had been labelled as functional until he himself persuaded a surgeon to explore the affected area. Accordingly, he was not readily disposed to regard his current symptoms as functional. In view of his declared weakness of muscles of articulation and of the legs, his colleague advised the exploration of the possibility that he was suffering from a myopathy such as myasthenia gravis. Since cases of the latter commonly respond to prostigmine, the subject agreed to test its action on himself.

3. The Intention

What began as an academic discussion of the psychological difficulties which may confront the thyroidectomized subject, if previously accustomed to long hours of intense intellectual work and with little capacity for relaxation of mental activity, thus encompassed both the possibility that the subject's condition might be in large measure attributable to involutional phenomena not directly connected with surgical treatment and the possibility that the subject (H) had a myopathy which might respond to prostigmine. It was the former consideration which led the investigator (S) to include in the treatment schedule both a *placebo* and *d*-amphetamine, but without the subject's knowledge before the completion of the trial.

It would be untrue to say that the writers were fully alert to all the issues stated in the foregoing introductory remarks when they undertook the experiment which follows. Nor would it be true to say that their intention was exclusively directed to evaluate a treatment, to throw light on a pathological condition, or (still less) to explore the usefulness of a particular method of inquiry. The truth is that the motives of each were mixed. In different measure each had some concern for the subject's health and some for the possibility of refining the type of procedure best described as *treatment under investigation*. The fact that one of the participants, the subject, had a special interest in the design of medical records and that each of them recognized at the outset the pitfalls which beset interpretation of a trial when the yardstick of efficacy is, in part, the affective testimony of the subject did, however, lead at the outset to the adoption of a regimen from the outcome of which they believe it to be possible to infer some useful indications concerning the proper conduct of the self-controlled and self-recorded trial.

Indeed, initial lack of premeditation with that end in view and inadequacy of preliminary planning as seen in retrospect have made them more alert to appropriate safeguards. Thus it goes without saying that the experiment recorded is not a model for others to follow. On the contrary, defects of design, as they came into focus *pari passu*, are the raw materials from which, as the writers hope, it is now possible to prescribe a more exacting regimen which may prove to be of great value.

4. Procedure

In so far as the undertaking had an aim, its aim was to ascertain whether physical fatigue and muscular weakness of which the subject complained was attributable to myasthenia *sensu stricto*. Thus the subject was aware that the experiment would entail the administration of prostigmine and a control substance, not necessarily inert. He was not aware that his colleage had included in the schedule of treatment courses both a placebo (*lactose*) and an active reagent (d-*amphetamine*). The self-record of his reaction to the latter is therefore an internal check on the reliability of the subject's testimony. To keep him wholly in ignorance both of the expected outcome of the treatment administered on any particular day and of the identity of the treatment administered on consecutive days, his colleague prescribed the channel of transmission as follows:

(1) The *investigator* (S) specified the daily regimen of dosage in conformity with a key identifying that of three consecutive days by a letter of the alphabet.

(2) The *liaison officer*, a colleague (C), transmitted the daily dosage to the patient in accordance with instructions referable to the code signs only, having therefore no means of inferring whether treatments on successive 3-day periods of a 26-fold sequence were identical, or of identifying the treatment on any single day.

(3) The *subject* (H) received the dosage from (C) without access to the code sign referable thereto.

Each dose (3 times daily) was a tablet of the same size to be swallowed with water. Thus the subject's only means of associating the experience of particular days with a particular treatment was the taste and/or texture of the tablet, if noticed. The subject reported no impression of such differences; and we may here discount this source of leakage. Ideally, it is easy to forestall in a comparable situation by recourse to a standard gelatine capsule.

As stated, the temporal unit of treatment by any one of the three substances administered was 3 days, on each of which the subject received three doses at 4-hourly intervals, the single doses being 5 mg. for amphetamine and 15 mg. for prostigmine. The experiment went on for 78 ($=32\times 6$) days covering the complete alphabetic code, the actual sequence of 3-day courses being: L. A. L. P. L. A. P. A. L. P. A. L. P. L. L. A. P. L. P. A. P. A. P. A. L. L. Thus the patient received the control agent ten times, prostigmine eight times, and amphetamine eight times. The intercalation of the control in parts of the cycle once or twice between the other two in succession or between successive administration of either is one safeguard against the danger of misinterpreting delayed action. In addition, the comparison of response on the first and third day of each 3-day course allows for a check which would not be applicable in an experiment involving 1-day courses only.

At the start the subject wrote a summary of normal daily routine of diet, motions, sleep, work, and other particulars referable to the immediate period antecedent to the beginning of the experiment and deemed to be relevant to the definition of the base-line reaction to L. Thereafter for each day, he completed a report recording his experience relative to the same and his affective reactions on rising, etc. He followed a consistent plan, though without a *pro forma* schedule, more by habit than by premeditation. Even so, the outcome illustrates forcibly the loss of information entailed by record-keeping in diary form.

The prescribed times for the three daily doses were 8.30 a.m., noon, and 5.30 p.m. The reader will note that the last dose of the day was not administered immediately before retiring. This was intentional on the part of the colleague (S) responsible for the treatment schedule. Late administration might have led the subject to infer that one of the agents was a soporific or the converse; but the diary discloses no comments on the subject's sleep experience other than times of retiring and rising.

5. Interpretation of the Record

The subject relied on his own experience to record his reactions during the course of the trial; and the testimony placed on record is classifiable under ten headings as below. To interpret the testimony recorded in diary form, it will suffice to adopt a 3-point scale:

Base-line Reaction (−) *i.e.* normal condition of subject immediately before experiment began.
No Reaction Stated (0).
Reinforced (*beneficial*) *Reaction* (+).

Item	Testimony−	Testimony+
I. } Motions	⩾3	<3
II. } Motions	Fluid (*at least once*)	Firm (*consistently*)
III. Mouth	Moist	Dry
IV. Rising	After 8.0 a.m.	Before 8.0 a.m.
V. Retiring	Before 11.0 p.m.	After 11.0 p.m.
VI. Work routine	No work after 6.0 p.m.	Work after dinner
VII. Physical activity	Minimal	Additional
VIII. Prevailing mood	Lethargic, depressed	Energetic, fresh, cheerful
IX. Waking reaction	Sluggish, depressed	Lively, cheerful
X. Appetite	Poor	Better than normal

Three items here call for special comment. The subject had recently had denture difficulties associated with buccal dryness and was moreover aware of the fact that one drug administered was prostigmine. The definition of the base-line reaction in Item III is arbitrary, but worth recording because it provides an internal check on testimony value. The value of Item VII is limited by the fact that there was little occasion to record a positive reaction in the mid-week. Contrariwise, the subject drove 80 miles each way to his country cottage at week-ends when there was opportunity for gardening and walking. As regards Item X, it suffices to say that we deem any favourable comment in the case notes as positive.

We may summarize the outcome in two ways. First, as in the left hand (*unweighted total scores*) columns of Tables I and II (opposite), we may record on how many days the testimony is classifiable in terms of each of the three categories (negative, zero, positive) for each treatment. For a 3-day course our record might disclose the following picture:

Day of Course	Item I	II	III	IV	V	VI	VII	VIII	IX	X
1	+	+	−	−	−	+	0	−	0	0
2	0	+	+	+	−	0	0	−	0	−
3	−	0	−	+	0	−	+	+	0	0

The score for the single 3-day unit would then be:

Score	Item I	II	III	IV	V	VI	VII	VIII	IX	X
−	1	0	2	1	2	1	0	2	0	1
0	1	1	0	0	1	1	2	0	3	2
+	1	2	1	2	0	1	1	1	0	0

This method of presentation takes no cognizance of a progressive response during the three-day course and outlasting it. One way of doing so employed for the right hand (*weighted total scores*) columns of Tables I–II is to assign a score of 1, 2, 3 to the appropriate testimony on successive days instead of assigning unit scores to each as above. Thus for III above we shall assign 1 to the first and 3 to the third negative sign and 2 to the single positive one. The outcome for the single 3-day unit will accordingly be:

Score	Item I	II	III	IV	V	VI	VII	VIII	IX	X
−	1	0	4	1	3	3	0	3	0	2
0	2	3	0	0	0	2	0	0	6	4
+	3	3	2	5	3	1	6	3	00	0

The alternative methods of presentation adopted were suggested by the subject, the first stage being to complete a schedule specifying for each day of the trial the testimony as negative, zero, or positive for each of the ten criteria named. Each of the writers felt that his own inclination might prejudice the

TABLE I

Item	Score	Total Scores					
		Unweighted			Weighted		
		L	P	A	L	P	A
I	–	1·6	9	5	3·2	20	10
	0	20·0	13	17	40·0	25	33
	+	2·4	2	2	4·8	3	5
II	–	4·0	12	5	8·8	23	10
	0	3·2	4	3	7·2	8	6
	+	16·8	8	16	32·0	17	32
III	–	0	0	0	0	0	0
	0	20·8	18	19	40·8	36	37
	+	3·2	6	5	7·2	12	11
IV	–	4·8	3	2	8·8	6	5
	0	17·6	18	19	36·8	35	34
	+	1·6	3	3	2·4	7	9
V	–	8·0	11	4	16·0	20	10
	0	8·0	5	4	16·0	11	8
	+	8·0	8	16	16·0	17	30
VI	–	10·4	6	8	19·2	13	17
	0	1·6	4	1	4·0	9	2
	+	12·0	14	15	24·8	26	29
VII	–	4·0	4	7	8·8	8	14
	0	10·4	12	11	21·6	25	22
	+	9·6	8	6	17·6	15	12
VIII	–	2·4	8	1	4·0	15	3
	0	19·2	15	15	40·8	32	29
	+	2·4	1	8	3·2	1	16
IX	–	0·8	0	1	2·4	0	1
	0	22·4	24	19	43·2	48	35
	+	0·8	0	4	2·4	0	12
X	–	2·4	1	0	4·8	1	0
	0	15·2	18	21	28·8	36	41
	+	6·4	5	3	14·4	11	7
TOTAL	–	38·4	54	33	76·0	106	70
	0	138·4	131	129	279·2	265	247
	+	63·2	55	78	124·8	109	163

TABLE II

Item	Score	Total Scores					
		Unweighted			Weighted		
		L	P	A	L	P	A
I	–	2·4	9	5	4·8	20	10
	0	19·2	13	16	38·4	25	32
	+	2·4	2	3	4·8	3	6
II	–	4·8	12	5	9·6	23	10
	0	4·8	4	3	11·2	8	6
	+	14·4	8	16	27·2	17	32
III	–	0	0	0	0	0	0
	0	21·6	18	19	43·2	36	37
	+	2·4	6	5	4·8	12	11
IV	–	4·8	3	5	8·8	6	13
	0	17·6	18	16	36·8	35	26
	+	1·6	3	3	2·4	7	9
V	–	8·0	11	4	16·0	20	10
	0	8·0	5	5	16·0	11	11
	+	8·0	8	15	16·0	17	27
VI	–	6·4	6	5	12·8	14	9
	0	2·4	1	2	4·8	2	5
	+	15·2	17	17	30·8	32	34
VII	–	0·8	0	1	2·4	0	1
	0	20·8	15	18	40·8	31	35
	+	2·4	9	5	4·8	17	12
VIII	–	2·4	12	2	4·0	23	4
	0	19·2	11	14	40·8	24	28
	+	2·4	1	8	3·2	1	16
IX	–	0·8	0	2	2·4	0	3
	0	21·6	23	16	43·2	45	30
	+	1·6	1	6	2·4	3	15
X	–	2·4	2	0	4·0	3	0
	0	13·6	16	19	28·0	31	38
	+	8·0	6	5	16·0	14	10
TOTAL	–	32·8	55	29	64·8	109	60
	0	148·8	124	128	303·2	248	248
	+	58·4	61	83	112·4	123	172

specification at this stage. Accordingly, they delegated it with appropriate instructions to two colleagues*, who devised separate presentations of the results (Tables I and II). The only noteworthy discrepancy refers to Item VII. This is due to the fact that one of the assessors interpreted driving a car in the week-end to the country cottage mentioned above as a positive response, and the other did not do so.

Since the subject received the *placebo* on 30 days and each of the alternative active agents on 24 days, the scores of the columns marked L in Tables I and II have been adjusted appropriately in the ratio 4 : 5, whence the decimals. Of the two methods of scoring exhibited, we may say that one will disclose a conservative picture of a progressive effect, the other may give undue weight to the terminal day of the course. In so far as the testimony of the two methods points to the same conclusion, as is here true, they reinforce one another.

* Our thanks are due to Dr. K. S. Jones of the Department of Clinical Psychiatry, United Birmingham Hospitals, and to Dr. K. W. Cross of the Department of Medical Statistics for undertaking this task.

In thus tabulating the scores it is necessary to take cognizance of the fact that Items IV (*Time of Rising*) and IX (*Waking Reaction*) refer to reactions antecedent to the stimulus prescribed for the day. Accordingly, we must antedate by one day the score recorded for each. It is for this reason that we cite them in one and the same column (19) for convenience of sorting in the illustrative predesigned proforma below.

6. NEED FOR PRE-DESIGNED RECORD

We need not here discuss in detail the positive outcome of the experiment recorded in the two Tables. The results, such as they are, give no indication of a myasthenia responsive to prostigmine, and the subject's reaction to amphetamine is consistent with what we might expect, if his symptoms were attributable to a functional disorder. Our concern is mainly with the defects which the record reveals. From this viewpoint, the outstanding fact disclosed is the *loss of information* attributable to

reliance on a case history recorded in diary form. There is an overall percentage of more than fifty unspecified responses. This illustrates a general principle of documentation more honoured in the breach than in the observance. Conclusive interpretation of medical documents is often impossible, because the observer records an event only if it forces itself on his attention. No record is wholly adequate unless it evokes a decisive answer to every question relevant to the enquiry it subserves.

A record *programmed* in accordance with this principle with a view to unequivocal instructions for hand or pre-coded mechanical sorting would have set out the ten items extracted from the case history in a daily *pro forma* as in cols. 17–21 of the illustrative specimen shown in Fig. 1 (opposite). For reasons already stated, it should also accommodate additional information with a view to:

(*a*) internal checks on the reliability of the subject's testimony;

(*b*) fruitful comparison of the results of similar experiments on other individuals.

Needless to say, no simple formula embraces the requirements of experiments of this type; but it may be of help to others, if we here proffer a layout which would have ensured a more satisfactory outcome of the experiment recorded in Section 5 above. Our concern with information of the sort specified by (*b*) above is to seek indications of physical build or case history likely to throw light on why some individuals respond in one way, others in another, to one and the same treatment. To accommodate such information it is desirable to complete separately:

(*i*) an *initial* record for rendition by *the investigator* at the beginning of treatment;

(*ii*) the *daily* record for rendition by *the subject*.

We discuss the layout of (*i*) in Section 7 below. In Fig. 1 we exhibit illustratively a pattern of the layout of the daily record, preferably for *printing on card* to facilitate hand sorting. To accommodate additional checks on the reliability of the subject's testimony, it includes several items for which the diary record made no provision.

7. Assessment of Individual Variation

In the introductory remarks of this communication, we have raised the issue: does the assessment of group response to a therapeutic agent by reliance on significance tests or on interval estimation give due weight to relevant information about individual variation contributory to response to treatment? The question is not new. In essence, the pattern of statistical procedure which became fashionable after the publication of Greenwood and Yule (1915) dates from the context of Quetelet's *Essai de physique sociale* (1835). This doubtless inspired Jules Gavarret's *Principes généraux de statistique médicale* (1840). Gavarret's plea for the statistical approach evoked little positive response at the time; but we may surmise that it inspired a rejoinder too little quoted of late years. In his lectures (published in English in 1927 as *An Introduction to the Study of Experimental Medicine*), Claude Bernard (1865) thus expressed the authentic reaction of the experimentalist to statistics conceived in Quetelet's terms as the science of averages:

> By destroying the biological character of phenomena, the use of *averages* in physiology and medicine usually gives only apparent accuracy to the results. From our point of view, we may distinguish between several kinds of averages: physical averages, chemical averages, and physiological and pathological averages. If, for instance, we observe the number of pulsations and the degree of blood pressure by means of the oscillations of a manometer throughout one day, and if we take the average of all our figures to get the true or average blood pressure and to learn the true or average number of pulsations, we shall simply have wrong numbers. In fact, the pulse decreases in number and intensity when we are fasting and increases during digestion or under different influences of movement and rest; all the biological characteristics of the phenomena disappear in the average. Chemical averages are also often used. If we collect a man's urine to analyse the average, we get an analysis of a urine which simply does not exist; for urine, when fasting, is different from urine during digestion. A startling instance of this kind was invented by a physiologist who took urine from a railroad station urinal where people of all nations passed, and who believed he could thus present an analysis of *average* European urine! Aside from physical and chemical, there are physiological averages, or what we might call average descriptions of phenomena, which are even more false. Let me assume that a physician collects a great many individual observations of a disease and that he makes an average description of symptoms observed in the individual cases; he will thus have a description that will never be matched in nature. So in physiology, we must never make average descriptions of experiments, because the true relations of phenomena disappear in the average; when dealing with complex and variable experiments, we must study their various circumstances, and then present our most perfect experiments as a type, which, however, still stands for true facts. In the cases just considered, averages must therefore be rejected, because they confuse, while aiming to unify, and distort while aiming to simplify. Averages are applicable only to reducing very slightly varying numerical data about clearly defined and *absolutely simple cases* . . . I acknowledge my inability to understand why results taken from statistics are called *laws;* for in my opinion

DAILY RECORD

Complete items specified by columns 11–12, 19, 23, 24, **before breakfast,** *items specified by 13–14, 23, 24,* **at 12.30 p.m.,** *items specified by 15–16, 23, 24* **at 10.00 p.m.,** *otherwise* **immediately** *before retiring.*

1–8 SUBJECT'S REGISTRATION No. (*Birmingham Code*) | | | | | | | | | | |

9–10 DAY OF TRIAL (*the first, second . . . 12th, etc. being 01, 02 . . . 12, etc.*) | |

11–12 PULSE RATE ON WAKING | |

13–14 PULSE RATE AT 12.30 p.m. | |

15–16 PULSE RATE AT 10.0 p.m. | |

17 MOTIONS *Insert number*

Firm	Loose	Fluid	Total

18 APPETITE *Ring appropriate item*

Appetite	Mouth Dry	Mouth Normal
Above normal	1	4
Normal	2	5
Poor	3	6

19 WAKING REACTION *Ring appropriate item*

Waking Reaction	Rising at or before 8.0 a.m.	Rising after 8.0 a.m.
Sluggish, depressed	1	4
Indifferent	2	5
Lively	3	6

20 WORK ROUTINE *Ring appropriate item*

Work Routine	Retired at or before 11.0 p.m.	Retired after 11.0 p.m.
None after 6.0 p.m.	1	3
Work after evening meal	2	4

21 PREVAILING MOOD
Ring appropriate item in **both** *A and B*

Appetite	A.—Prevailing Mood			B.—Physical Activity		
	Lethargic, depressed	Indifferent	Energetic, cheerful	Above normal	Normal	Little
Good	1	7	13	2	8	14
Indifferent	3	9	15	4	10	16
Poor	5	11	17	6	12	18

22 SLEEP *Ring appropriate item*

1. Steady	2. Fitful or Little

23 SYSTOLIC B.P. *Insert number*

Before Breakfast			12.30 p.m.			10.0 p.m.		

24 DIASTOLIC B.P. *Insert number*

Before Breakfast			12.30 p.m.			10.0 p.m.		

TREATMENT (*To be filled in by the Investigator*)

FIG. 1

> scientific law can be based only on certainty, on absolute determinism, not on probability . . . In every science, we must recognize two classes of phenomena; first, those whose cause is already defined; next, those whose cause is still undefined. With phenomena whose cause is defined, statistics have nothing to do; they would even be absurd. As soon as the circumstances of an experiment are well known, we stop gathering statistics: we should not gather cases to learn how often water is made of oxygen and hydrogen; or when cutting the sciatic nerve, to learn how often the muscles to which it leads will be paralysed. The effect will occur always without exception, because the cause of the phenomena is accurately defined . . . Certain experimenters, as we shall later see, have published experiments by which they found that the anterior spinal roots are insensitive; other experimenters have published experiments by which they found that the same roots were sensitive. These cases seemed as comparable as possible; here was the same operation done by the same method on the same spinal roots. Should we therefore have counted the positive and negative cases and said: the law is that anterior roots are sensitive, for instance, 25 times out of a 100? Or should we have admitted, according to the theory called the law of large numbers, that in an immense number of experiments we should find the roots equally often sensitive and insensitive? Such statistics would be ridiculous, for there is a reason for the roots being insensitive and another reason for their being sensitive; this reason had to be defined; I looked for it, and I found it; so that we can now say: the spinal roots are always sensitive in given conditions, and always insensitive in other equally definite conditions.

Many of our contemporaries will brush aside the claim that scientific law must be based on certainty by citing the principle of uncertainty in electronics; but the use of statistical theory to construct hypotheses which stand or fall by the arbitrament of experiment in no sense conflicts with the view Claude Bernard here asserts. If study of the behaviour of an organism leads us to proffer an average as the outcome of prescribed conditions, we have not as yet established the law of its behaviour. We have done so when, and only when, a more refined prescription of the conditions will ensure a uniform response. How far statistical methods can indeed contribute to the initial stage of an investigation by screening likely clues is not the issue which Claude Bernard raises. In contemporary terms what he rightly rejects is the now widely current practice of publishing as a discovery what is at best an encouragement to further examination.

If the comments of Claude Bernard on the use of averages were salutary in his time, they are still more so in our own context. In Bernard's day, the statistician was content to assert a modest claim to the supervision of interpretation; in ours, he claims the prerogative of prescribing the "Design of Experiments", *i.e.* the conduct of experiments to supply figures which certain test and estimation procedures can, or purport to be able to, accommodate. Indeed, Cattell and Williams (1953) referring to the use of factor analysis in physiological enquiry, go so far as to say:

> Although it is true that any experimental statistical design permits one to look for a law in the relation between certain variables, without entering the experiment with too definite a hypothesis as to what form the law must take, *factor analysis is unique in demanding no prior hypothesis and in being automatically productive of a hypothesis.* One *may* start with a highly specific hypothesis about the forms and relations of the factors, but this is not necessary. For a pattern of factor loadings—a cluster of variables—suggests immediately something about the nature of a certain influence behind them. This creates the hypothesis by which one enters the next experiment into which some new variable can now be inserted and which may be designed more explicitly to measure the hypothesized influence. But, in entering the initial factor analytic study, one need not have any more definite a hypothesis than is necessary to choose a set of variables, the relations among which are likely to be of interest, in this case to human physiology. (*Italics inserted*)

Though the acceptance of such claims is a repudiation of the methods by which the experimental sciences have attained their present status, no experimental scientist of Bernard's standing has hitherto accepted the challenge publicly. It is therefore pertinent to state that the issue is essentially a matter of method on which mathematicians as such have no prior claim to arbitrate. If, as we so often hear, statistics is the science of averages, the statistician has a special claim to our attention when the end in view is to record averages, as is true of demographic studies and administrative inquiries which do not concern themselves with individuals as such; but it is for the biologist and the clinician themselves to decide whether an average can be a satisfactory answer to questions they ask about the *individual* organism. The statement that solutions of iodine in KI makes starch paste blue in $75 \pm 1 \cdot 5$ per cent. of samples examined will not commend itself to a chemist. He will wish to know what impurities, or what range of *p*H values, etc., determine when the reaction does or does not occur. Such has been the attitude in which physiologists have hitherto undertaken the investigation of animal behaviour. If we abandon it, we are lowering our standards and concealing our retreat from a position of hard-won advantage behind an impressive façade of irrelevant algebra.

To appreciate the full force of the contention cited above, we shall do well to recall the history of the therapeutic trial. The association of stochastic theory with medicine antedates Gavarret by three-quarters of a century. About the middle of the 18th century, when the Turkish practice of vaccination had lately come into Western Europe, the statistics of smallpox enlisted the interest of both Jean d'Alembert and Daniel Bernoulli. By a method which can stand on its feet without recourse to the classical theory of probability the latter was able to show how the incidence of a disease which confers immunity must decline as age advances; but the mathematical viewpoint made little headway in medicine till smallpox became an issue for legislative intervention. Indeed, public controversy concerning compulsory immunization in the closing years of the 19th and in the first few decades of the 20th century sufficiently explains the eagerness to invoke the statistician as arbitrator of the claims of the laboratory and of the layman.

Henceforth, we have been more and more disposed to assume that the problem of the prophylactic and that of the therapeutic trial are formally identical. This identification is admissible only if we lose sight of the end in view. In the practice of preventive medicine our concern is with numbers, and the framework of our problem is essentially one of social accountancy. In the practice of curative medicine our concern is with the sick individual, and reliance on averages as a criterion of preference may lead us to recommendations inconsistent with the end in view.

That this is so will be immediately apparent, if we pause to consider a fictitious situation. We may suppose:

(*i*) that a Disease D is incurable if untreated;

(*ii*) that a clinical trial of the usual type leads us to assess the recovery-rate under Treatment A as about 25 per cent. and the recovery-rate under Treatment B as about 50 per cent.

In such circumstances, we too easily then content ourselves with a recommendation to step up the recovery rate by 25 per cent. by substituting Treatment B for Treatment A. If so, our preoccupation with averages has blinded us to biological realities. Were we alert to the manifold interaction of nature and nurture, the outcome would invite us to ask the question: what peculiarities are common to individuals who respectively respond or fail to respond to one or other treatment?

For heuristic reasons, let us now assume that:

(*a*) persons with grey or brown eyes invariably respond to Treatment B and fail to respond to Treatment A;

(*b*) persons with blue eyes invariably respond to Treatment A but not to Treatment B;

(*c*) blue-eyed individuals and individuals with grey or brown eyes occur in the population in the ratio 25 : 50;

(*d*) our clinical trial groups are representative in the sense that the ratio of blue-eyed persons to persons with grey or brown eyes is also close to 25 : 50.

On these assumptions the recovery rate would be 100 per cent. if all D patients with blue eyes continued to receive Treatment A and all D patients with gray or brown eyes henceforth received Treatment B.

The advantage of the procedure outlined in this communication is that it leaves us free to *select* for inquiry individuals with characteristics putatively relevant to the fullest use of an alternative procedure, inasmuch as:

(*a*) clinical experience already suggests what peculiarities of the individual are indicative of favourable prognosis or otherwise;

(*b*) intelligently comprehensive design of the *initial record* accommodates a range of variables likely to screen such characteristics, if such there be.

With this end in view, it is not desirable to proscribe the inclusion of a narrative *case history* of organic and functional disorder in a folder for each subject, though a general *pro forma* such as the *Cornell Medical Index Health Questionnaire* has much to commend it. In any event, it is highly desirable to make provision for one or more predesigned and pre-coded documents recording:

(*a*) physique;

(*b*) functional state assessed by biochemical and biophysical tests;

(*c*) personality structure.

Of (*c*) it suffices to say that the writers are cooperating in an experimental inquiry with reference to validification of appropriate descriptive nomenclature, and that no satisfactory pre-design for the documentation of basic data of interest to the experimental psychiatrist is likely to emerge unless it is possible to reach agreement about the vocabulary of what Woodger (1952) calls the *observational record.* As regards (*b*), a prerequisite to the undertaking is the availability of suitable codes; and it suffices to remark that the Birmingham Department of Medical Statistics will shortly publish such codes in an exposition of the system of self-coded documents already on trial in the professorial units of the Birmingham Medical School. For illustrative purposes, we none the less venture to proffer a specimen *pro forma* for recording anthropometric data in the initial record for rendition by the

investigator (Figs 2 and 3). The lay-out shown is suitable for the Powers-Samas machinery (65-column punch card, accommodating 39 items of information per column).

8. Summary

(1) The current prescription for the conduct of the *Clinical Trial* relies on statistical assessment of the response of treated and untreated individuals chosen randomwise. The credentials of current procedures for statistical validification of the outcome are by no means immune to criticism; but the comparison of treatment groups in terms of averages is open to criticism for other reasons, in particular because the demonstration that one treatment has a higher recovery rate than another encourages us to evade the question: why do some individuals respond or fail to respond to one or other treatment? More explicitly, we may frame the same question as follows: what circumstances of nature or nurture determine whether the individual patient will or will not respond to such and such a treatment? If an answer to this is indeed the goal of inquiry, the outcome of subjecting individuals with some characteristics to Treatment A and others to Treatment B may well prove to be more advantageous than substituting for Treatment A another Treatment B with a higher average recovery rate.

(2) This communication explores the design of a trial which has as its aim to assess the response of a given individual with explicitly specifiable characteristics to alternative treatments in conformity with the customary conduct of pharmacological inquiry on individual animal subjects. Its aim is to clarify what conditions we must impose on such a trial if it is to conform to the traditional canons of pharmacological experiments, here described as *self-controlled*, and to advance suggestions for recording reliable information in the most economical way. By *self-controlled* in this context we connote:

(*i*) the procedure of repeated administration of alternative stimuli to one and the same individual;

(*ii*) reliance on one-to-one correspondence of stimulus and response to validate our conclusions.

Since all experimental biologists recognize that physiology has advanced conspicuously by recourse to such procedures without reliance on statistical assessment of group averages, the only debatable issues are:

(*a*) how far it is possible to standardize the conditions of observation in conformity with the customary safeguards imposed on the conduct of a comparable experiment on an animal subject;

(*b*) whether the effort necessarily expended in doing so limits the usefulness of the recipe to situations in which the investigator has sufficient time and facilities for direct observation of the subject.

(3) As regards 2(*a*) above, the major desideratum is that the subject should remain ignorant of the sequence of presentation. Where the stimulus is a drug, this is achievable by:

(*i*) administration of the drug itself in appropriate disguise, *e.g.* capsule or suppository;

(*ii*) defining the instructions for the course in terms which disclose no clue to the identity of the stimulus.

The second of these is easy to accomplish if an intermediary transmits the stimulus in accordance with a code to which he or she has no access and the subject receives the dose without disclosure of the code sign.

(4) As regards 2(*b*) above, it is our submission that it is possible to prescribe a suitable form of daily self-coded record which the subject can complete *pari passu* with the sequence of presentation. If a self-controlled experiment may thus be also and reliably *self-recorded*, we can envisage the conduct on a wide front of researches of a type not hitherto undertaken. Desiderata for successful use of this procedure are that the record should:

(*a*) provide means of checking the reliability of the subject's testimony;

(*b*) invite verbal responses which are not ambiguous.

Where it is possible to pair off co-habitant subjects, reciprocal comparison of the subject's testimony to his or her own overt behaviour by the co-subject may both provide an additional means of validating the testimony of the record and of clarifying the interpretation of verbal responses relevant to psychiatric diagnosis.

(5) Needless to say this preliminary prescription of a self-controlled and self-recorded trial is referable to *low-grade morbidity* alone. For acute and dangerous conditions it can offer nothing; but its usefulness is not negligible on that account. As never before, curative medicine has now to take cognizance of the prominent role of the so-called psychosomatic disorders in the day to day work of general practice and of the large field of mental sickness which lies outside existing provisions for institutional care.

INITIAL INDIVIDUAL RECORD

(for completion by the investigator or qualified assistant)

PART I. ANTHROPOMETRIC DATA (a) Males

1–8 SUBJECT'S REGISTRATION No. (*Birmingham Code*) [| | | | | | | | | |]

9–12 DATE OF BEGINNING OF TREATMENT

Day	Month	Year (19XY)
01–31	01–12	XY

13–14 STANDING HEIGHT (*cm.*) [| |]

15–16 SITTING HEIGHT (*cm.*) [| |]

17–18 WEIGHT (*lb.*) [| |]

19–20 MAXIMUM THORACIC CIRCUMFERENCE (*cm.*) (at nipple line) [| |]

21–22 MINIMUM THORACIC CIRCUMFERENCE (*cm.*) (at nipple line) [| |]

23–24 MAXIMUM TRANSVERSE CHEST DIAMETER (*cm.*) (at nipple line) [|]

25–26 MINIMUM TRANSVERSE CHEST DIAMETER (*cm.*) (at nipple line) [|]

27–28 ABDOMINAL CIRCUMFERENCE (*cm.*) (at umbilicus) [| |]

29 COSTAL ANGLE (*degrees*) AT INSPIRATION

	<80	80–84	85–89	90–94	95–99	100–104	105–109	110–114	115–119	120 and over
Code . .	0	1	2	3	4	5	6	7	8	9

30 COSTAL ANGLE (*degrees*) AT EXPIRATION

	<80	80–84	85–89	90–94	95–99	100–104	105–109	110–114	115–119	120 and over
Code . .	0	1	2	3	4	5	6	7	8	9

31–32 LEG LENGTH (*cm.*) (ant. sup. iliac spine to tip of lateral malleolus) [| |]

33–34 ARM LENGTH (*cm.*) (acromion to first carpal crease) [|]

35 HAND LENGTH (*cm.*) (first carpal crease to tip of middle finger) [|]

36–37 BISACROMIAL WIDTH OF SHOULDERS (*cm.*) [|]

38 BICRISTAL PELVIC WIDTH (*cm.*) [|]

FIG. 2

PART I. ANTHROPOMETRIC DATA (a) Males—*cont.*

39 SACRO-PUBIC DEPTH (*cm.*) ☐☐

40–41 NECK GIRTH (*cm.*) (at upper level of thyroid cartilage) ☐☐

42 MAXIMUM CEPHALIC BREADTH (*cm.*) ☐☐

43 MAXIMUM CEPHALIC LENGTH (*cm.*) ☐☐

44–45 MAXIMUM TRUNK LENGTH (*cm.*) (suprasternal notch to pubic symphysis) ☐☐

46–47 MINIMUM TRUNK LENGTH (*cm.*) (suprasternal notch to pubic symphysis) ☐☐

48–49 MEAN (*Right–Left*) MAXIMUM GIRTH OF CALF (*cm.*) ☐☐

50 SEXUAL DEVELOPMENT

	Voice Infantile		
	Both testicles retractile	One testicle only retractile	Both testicles non-retractile
Code ..	0	1	2

	Voice Breaking		
	Both testicles retractile	One testicle only retractile	Both testicles non-retractile
Code ..	3	4	5

	Voice Adult		
	Both testicles retractile	One testicle only retractile	Both testicles non-retractile
Code ..	6	7	8

51 PUBIC—AXILLARY HAIR

	Pubic Hair Absent		Pubic Hair Present	
	Axillary hair absent	Axillary hair present	Axillary hair absent	Axillary hair present
Code ..	0	1	2	3

52 HAIR OF SCALP

White	Grey	Yellow	Red	Light Brown	Dark Brown	Black
1	2	3	4	5	6	7

53 HAIR OF CHIN

White	Grey	Yellow	Red	Light Brown	Dark Brown	Black
1	2	3	4	5	6	7

54 HAIR OF UPPER LIP

White	Grey	Yellow	Red	Light Brown	Dark Brown	Black
1	2	3	4	5	6	7

FIG. 2—*continued*

PART I. ANTHROPOMETRIC DATA (a) Males—*cont.*

55 HAIR OF CHEST

White	Grey	Yellow	Red	Light Brown	Dark Brown	Black
1	2	3	4	5	6	7

56 AXILLARY HAIR

White	Grey	Yellow	Red	Light Brown	Dark Brown	Black
1	2	3	4	5	6	7

57 PUBIC HAIR

White	Grey	Yellow	Red	Light Brown	Dark Brown	Black
1	2	3	4	5	6	7

58 EYE COLOUR

Pink	Blue	Grey	Green	Hazel	Dark
1	2	3	4	5	6

59 ISO-AGGLUTININ GROUP

A	B	O	AB
1	2	3	4

60 RHESUS (D) AND M-N GROUP

M		N		MN	
Rh+	Rh−	Rh+	Rh−	Rh+	Rh−
1	2	3	4	5	6

FIG. 2—*continued*

INITIAL INDIVIDUAL RECORD

for completion by the investigator or qualified assistant)

PART I. ANTHROPOMETRIC DATA (b) FEMALES

1–8 SUBJECT'S REGISTRATION No. (*Birmingham Code*) | | | | | | | | | | |

9–12 DATE OF BEGINNING OF TREATMENT

Day	Month	Year (19XY)
01–31	01–12	XY

13–14 STANDING HEIGHT (*cm.*) | | |

15–16 SITTING HEIGHT (*cm.*) | | |

17–18 WEIGHT (*lb.*) | | |

FIG. 3

PART I. ANTHROPOMETRIC DATA (b) Females—*cont.*

19–20 MAXIMUM THORACIC CIRCUMFERENCE (*cm.*) (at nipple line)

21–22 MINIMUM THORACIC CIRCUMFERENCE (*cm.*) (at nipple line)

23–24 MAXIMUM TRANSVERSE CHEST DIAMETER (*cm.*) (at nipple line)

25–26 MINIMUM TRANSVERSE CHEST DIAMETER (*cm.*) (at nipple line)

27–28 ABDOMINAL CIRCUMFERENCE (*cm.*) (at umbilicus)

29 COSTAL ANGLE (*degrees*) AT INSPIRATION

	<80	80–84	85–89	90–94	95–99	100–104	105–109	110–114	115–119	120 and over
Code ..	0	1	2	3	4	5	6	7	8	9

30 COSTAL ANGLE (*degrees*) AT EXPIRATION

	<80	80–84	85–89	90–94	95–99	100–104	105–109	110–114	115–119	120 and over
Code ..	0	1	2	3	4	5	6	7	8	9

31–32 LEG LENGTH (*cm.*) (ant. sup. iliac spine to tip of lateral malleolus)

33–34 ARM LENGTH (*cm.*) (acromion to first carpal crease)

35 HAND LENGTH (*cm.*) (first carpal crease to tip of middle finger)

36–37 BISACROMIAL WIDTH OF SHOULDERS (*cm.*)

38 BICRISTAL PELVIC WIDTH (*cm.*)

39 SACRO-PUBIC DEPTH (*cm.*)

40–41 NECK GIRTH (*cm.*) (at upper level of thyroid cartilage)

42 MAXIMUM CEPHALIC BREADTH (*cm.*)

43 MAXIMUM CEPHALIC LENGTH (*cm.*)

44–45 MAXIMUM TRUNK LENGTH (*cm.*) (suprasternal notch to pubic symphysis)

46–47 MINIMUM TRUNK LENGTH (*cm.*) (suprasternal notch to pubic symphysis)

48–49 MEAN (*Right–Left*) MAXIMUM GIRTH OF CALF (*cm.*)

50 SEXUAL DEVELOPMENT

	Before Menarche	After Menarche		
		Before Menopause	During Menopause	After Menopause
Code ..	0	1	2	3

FIG. 3—*continued*

PART I. ANTHROPOMETRIC DATA (b) Females—*cont.*

51 PUBIC—AXILLARY HAIR

	Pubic Hair Absent		Pubic Hair Present	
	Axillary hair absent	Axillary hair present	Axillary hair absent	Axillary hair present
Code ..	0	1	2	3

52–53 DATE OF 1st DAY OF LAST MENSES BEFORE ADMISSION

Day		Month	
01–31		01–12	

54–55 DATE OF 1st DAY OF FIRST MENSES AFTER ADMISSION

Day		Month	
01–31		01–12	

56–57 DATE OF 1st DAY OF SECOND MENSES AFTER ADMISSION

Day		Month	
01–31		01–12	

58 HAIR OF SCALP

White	Grey	Yellow	Red	Light Brown	Dark Brown	Black
1	2	3	4	5	6	7

59 AXILLARY HAIR

White	Grey	Yellow	Red	Light Brown	Dark Brown	Black
1	2	3	4	5	6	7

60 PUBIC HAIR

White	Grey	Yellow	Red	Light Brown	Dark Brown	Black
1	2	3	4	5	6	7

61 EYE COLOUR

Pink	Blue	Grey	Green	Hazel	Dark
1	2	3	4	5	6

62 ISO-AGGLUTININ GROUP

A	B	O	AB
1	2	3	4

63 RHESUS (D) and M-N GROUP

M		N		MN	
Rh+	Rh−	Rh+	Rh−	Rh+	Rh−
1	2	3	4	5	6

FIG. 3—*continued*

REFERENCES

Bernard, C. (1865). "Introduction à l'étude de la médecine expérimentale". Baillière, Paris. Eng. trans. by H. C. Greene (1927). "An Introduction to the Study of Experimental Medicine". Macmillan, New York.

Cattell, R. B., and Williams, H. F. V. M. (1935). *British Journal of Preventive and Social Medicine*, **7**, 141.

Gavarret, J. (1840). "Principes généraux de statistique médicale". Bechet and Labé, Paris.

Greenwood, M., and Yule, G. V. (1915). *Proc. roy. Soc. Med.*, **8**, 113.

Head, H., Rivers, W. H. R., Holmes, G., Sherren, J., Thompson, T., and Riddock, G. (1920). "Studies in Neurology". Oxford University Press, London.

Oliver, G., and Schafer, E. A. (1894–95). *J. Physiol.*, **16**, **17**, **18**.

Quetelet, A. (1835). "Essai de physique sociale". Bachelier, Paris.

Woodger, J. H. (1952). "Biology and Language". (Tarner Lectures, 1949–50). University Press, Cambridge.

Brit. J. prev. soc. Med. (1953), 180–193

HISTORY OF POLIOMYELITIS IN CORNWALL AND DEVON

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Introduction

Though it was begun quite independently, this paper may be regarded as an extension of the work of Benjamin and Logan (1953). They have studied the distribution of notifications of poliomyelitis in Great Britain as a whole, whereas we have examined a smaller area in detail.

The present study was undertaken for several reasons:

(*i*) Cornwall and Devon are pre-eminently the "holiday" counties of England, and are visited by large numbers every summer. Most of the visitors spend their holiday in a coastal resort but many stay inland in a farmhouse or camp. It seems reasonable to suppose that, if importation of infection is a potent factor in the origin of epidemics, the seaside towns should show some preponderance of cases over the years.

(*ii*) The two counties had one of the earliest severe epidemics of poliomyelitis recorded in England—that of 1911. In that year 154 cases were reported in the two admininistrative counties (Cornwall, 42; Devon, 108) up to September 16, and there were more later. In addition, some seventy cases were reported by Soltau (1911), in the towns which now make up the county borough of Plymouth. The distribution of cases up to September 16, 1911, in the two counties was reported in detail by Reece (1912) and the later cases were described by the County Medical Officers of Health for Cornwall and Devon. No cases were reported in the county borough of Exeter. Between 1919 and 1946 (Fig. 1) there appear to have been no generalized outbreaks, but there were some local ones which are of interest when considered in relation to what came before and after and to what was happening elsewhere in England. In 1947 there was a generalized epidemic in England as a whole and in both counties more cases than usual were reported, but on the whole, incidence was lower than might have been expected. After the general lull of 1948, however, the attack rates in 1949 and 1950 in both counties were higher than in England and Wales as a whole. (Table I, opposite.)

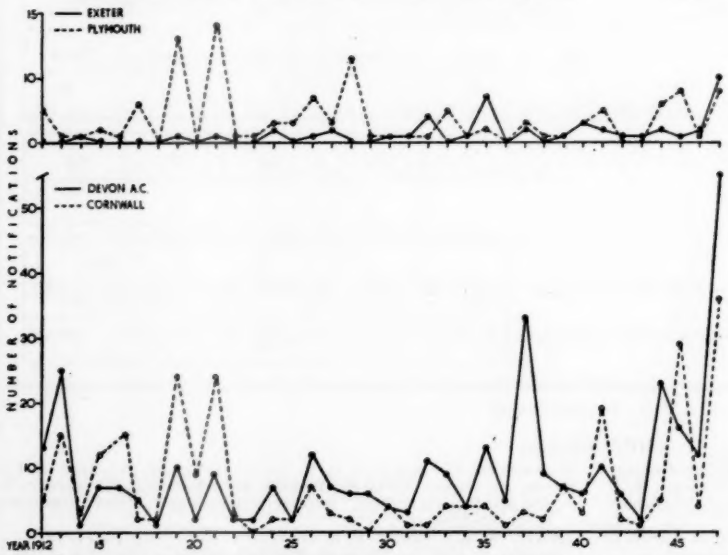

FIG. 1.—Annual notifications of poliomyelitis in Cornwall A.C., Devon A.C., Exeter C.B., and Plymouth C.B., 1912–47.

Fig. 1 suggests that there may have been some persistence of activity for several years after 1911 and some renewal of it before 1947. When compared with the true epidemic years, this activity was slight and has therefore passed unnoticed.

(*iii*) Cornwall and Devon have certain advantages over most other counties of England for the study of the distribution of disease. There is a reasonably sharp distinction between urban and rural

TABLE I

NOTIFICATION RATES OF POLIOMYELITIS PER 10,000 LIVING IN ENGLAND AND WALES, CORNWALL A.C., DEVON A.C., EXETER C.B., AND PLYMOUTH C.B., FOR INDIVIDUAL YEARS 1947–52, AND MEAN ANNUAL RATE FOR 6 YEARS 1947–52

Area	1947	1948	1949	1950	1951	1952	Mean Annual Rate 1947–52
England and Wales ..	1·8	0·4	1·4	1·8	0·6	0·9	1·1
Cornwall A.C.	1·1	0·6	3·5	2·9	1·1	0·6	1·6
Devon A.C.	1·1	0·4	1·8	3·5	1·0	1·2	1·5
Exeter C.B.	1·4	0·8	0·8	3·4	0·5	1·3	1·4
Plymouth C.B.	0·4	0·2	1·0	1·5	1·2	0·4	0·8

(N.B.—Rates for individual years calculated from the Registrar-General's estimated populations for each year, mean annual rates from populations given in the preliminary report on the 1951 census.)

conditions, lines of communications are not quite so complicated as in most other counties, and the holiday resorts may be distinguished from areas predominantly concerned with some local industry. Such distinctions are never absolute, but in these two counties they are fairly definite.

Unfortunately, information about the occurrence and distribution of cases of poliomyelitis is very unequal for the whole period, but from 1912–30 we have records of the cases reported in the two administrative counties and in the two county boroughs (Exeter and Plymouth). The records are summarized in Fig. 1. From 1931 onwards we have information about the number of cases reported each year in every sanitary district. For some years there is a little more, and for the years from 1947 considerably more, detailed information about distribution. In reviewing the whole story, one is struck by the frequency with which the disease seems to appear in almost the same, but not quite the same areas at intervals sometimes of many years. One is also struck by the comparatively small number of cases reported in the big seaside resorts like Torquay and Paignton, and by the fact that the outbreaks have tended to occur either in rather out-of-the-way parts like Holsworthy or in small industrial towns like Newton Abbot and St. Austell. It is convenient to divide the description into three parts:

(1) the epidemic of 1911,
(2) the inter-epidemic period, 1912–46,
(3) the recent epidemic period, 1947–52.

EPIDEMIC OF 1911

When we compare the epidemic of 1911 with more recent experience we have to make certain allowances. Although sporadic cases of the disease had been described for many years, epidemics were only just beginning in Great Britain. The districts in N.W. Devon and N.E. Cornwall, which were chiefly affected, must then have been a great deal more remote than they are now, and even to-day Holsworthy R.D., which had the highest attack rate of any county district, is very much off the beaten track. There was at that time no compulsory notification of poliomyelitis; in fact the events of 1911 here and elsewhere, were the chief reason for the introduction of compulsory notification. It therefore seems likely that, according to present-day standards, the reporting of cases was defective and that many cases which would now be counted as poliomyelitis were then overlooked. There is a little indirect evidence of this in the report of Reece (1912), for 34 of the 154 patients for whom he obtained details died. A case mortality rate of 22·1 per cent. is rather more than double that which is usual nowadays. Even if we take Reece's figures at their face value the attack rate in the Holsworthy R.D. (59·8 per 10,000) was extraordinarily high. Table II, modified from Reece's report, shows the distribution of the known cases by sanitary districts on a geographical basis with rates per 10,000 population for districts with five or more cases.

TABLE II

(Modified from Reece, 1912)

A.—PRINCIPAL FOCI IN N.W. DEVON AND N.E. CORNWALL

District	*Population*	*Cases*		*Rate per 10,000*
Holsworthy R.D.	7,022	42		59·8
Holsworthy U.D.	1,499	3		
Bideford R.D.	6,355	17		26·8
Stratton and Bude U.D. ..	2,976	8	79	26·9
Stratton R.D.	4,924	5		10·2
Torrington R.D.	8,970	2		
Northam U.D.	5,500	2		

B.—LESSER FOCI. 1. SOUTH MOLTON AND BARNSTAPLE

South Molton R.D.	10,908	8		7·3
South Molton U.D.	2,742	7	17	25·5
Barnstaple U.D.	14,485	1		
Barnstaple R.D.	18,229	1		

(*Stoke Rivers—see special note in text.*)

2. NEWTON ABBOT

Newton Abbot U.D.	13,711	4		
Newton Abbot R.D.	19,507	4		
Teignmouth U.D.	9,215	2	15	
Torquay U.D.	38,771	2		
Totnes R.D.	12,293	2		
Ashburton U.D.	2,494	1		

3. PENRYN, FALMOUTH, AND EAST KERRIER

Penryn M.B.	3,092	7		22·7
Falmouth M.B.	13,132	1		
East Kerrier R.D.*	8,009	7	24	8·8
Redruth R.D.*	18,002	3		
Camborne U.D.*	15,829	3		
Truro R.D.*	30,515	3		

C.—SCATTERED CASES

DEVON			CORNWALL		
Crediton R.D. ..	1		Bodmin R.D.	1	
Crediton U.D. ..	1		Launceston R.D. ..	3	
Exmouth U.D. ..	2		Launceston U.D. ..	1	9
Honiton R.D.	1	10	Newquay U.D. ..	2	
Tavistock R.D. ..	3		St. Austell R.D. ..	2	
Tavistock U.D. ..	1				
Tiverton R.D.	1				

* There have been considerable changes in Local Government Divisions in Cornwall since 1911.

Fig. 2 shows the geographical relationships of the administrative areas.

The first case in Devon was noted on May 3, 1911, in Holsworthy U.D., and the first in Cornwall on May 27, 1911, in Penryn U.D. The outbreak in Penryn was described by Gregor and Hopper (1911).

Some of these rates are very high and the only county district which has experienced a higher rate in recent years is the small town of Fowey, which had fifteen cases in 1950 (64 per 10,000).

Reece's account is incomplete in that it ends with the week ending September 16, 1911, when eleven cases were reported. There were more cases in both counties after Reece's departure, but those in Cornwall are of particular interest, because it is clear from the County Medical Officer's Report for 1911 that St. Austell U.D. and R.D. and Truro U.D. and R.D. were involved in the later stages of the epidemic. The later figures for these areas are:

St. Austell R.D., 13; U.D., 3. Truro U.D., 1; R.D., 5.

It is possible to reconstruct a general picture of the distribution of the known cases in the 1911 epidemic from Reece's report and the Annual Report of the County Medical Officers of Health for 1911 as follows:

The main focus was in Holsworthy R.D. with offshoots to the West at Bude and to the North in the parishes of Hartland and Clovelly (Fig. 3, opposite). Within Holsworthy R.D. cases were concentrated in a relatively small number of parishes:

(1) in Holsworthy itself, in the parishes of Thornbury and Cookbury to the north east and in Clawton and Pyworthy to the south;
(2) in W. Putford and Bradworthy.

No cases were reported from large areas of Holsworthy R.D. and several instances of multiple cases in one household were reported (see Fig. 3, inset).

The lesser foci were:

(*a*) South Molton with extension along the main road to Barnstaple and possibly to Stoke Rivers.*
(*b*) Penryn and East Kerrier with extension north to Camborne and possibly east to Truro and St. Austell though it is not clear whether St. Austell should be regarded as a separate focus.
(*c*) Newton Abbot and the South Devon coast towns of Exmouth, Teignmouth, Torquay, Totnes.

* Reece includes in his report (p. 42) an account of a localized outbreak of an illness resembling non-paralytic poliomyelitis in the village of Stoke Rivers (Barnstaple R.D.) between August 21 and September 30, 1911. In this village with a population of 119 no less than 36 persons had fairly definite attacks and nine others possible attacks. Reece's account has some similar features to that given by Hargreaves (1949) of the events at N. Tamerton in July, 1947, though there three paralytic cases occurred.

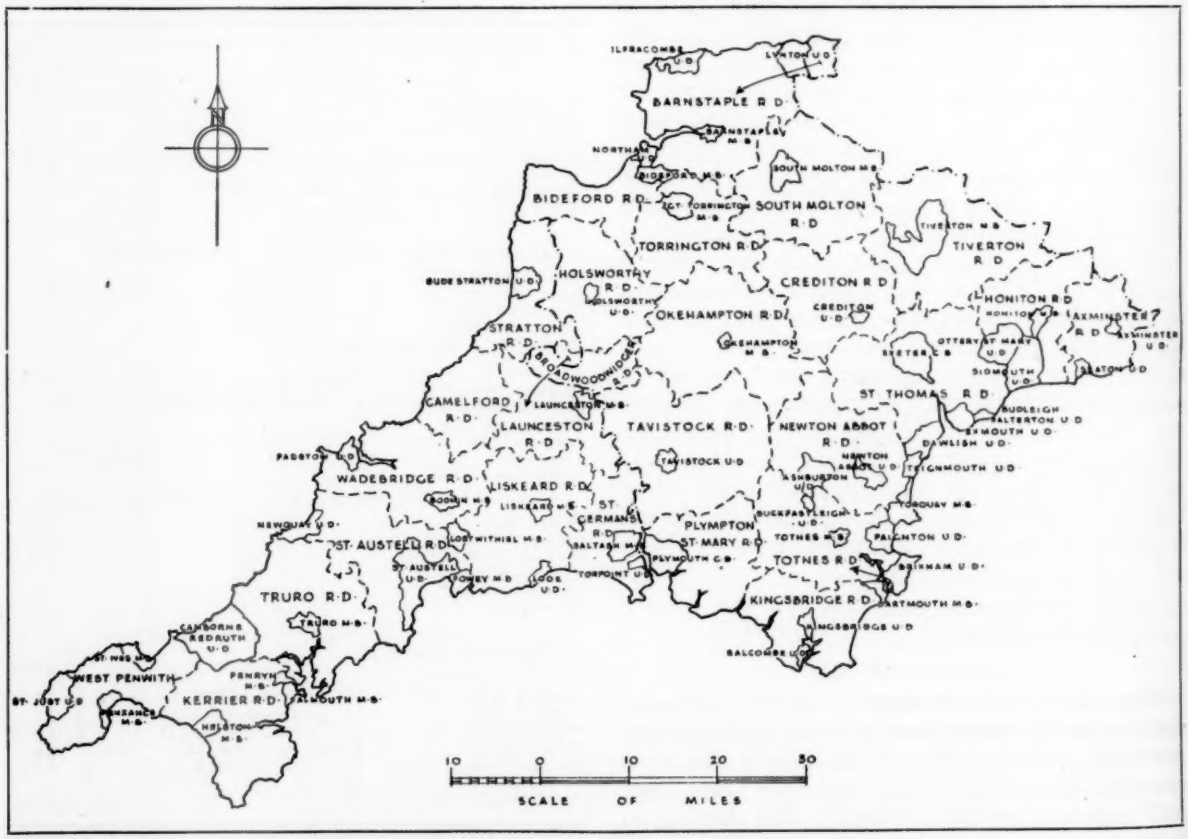

FIG. 2.—Administrative areas of Cornwall and Devon (Key Map).

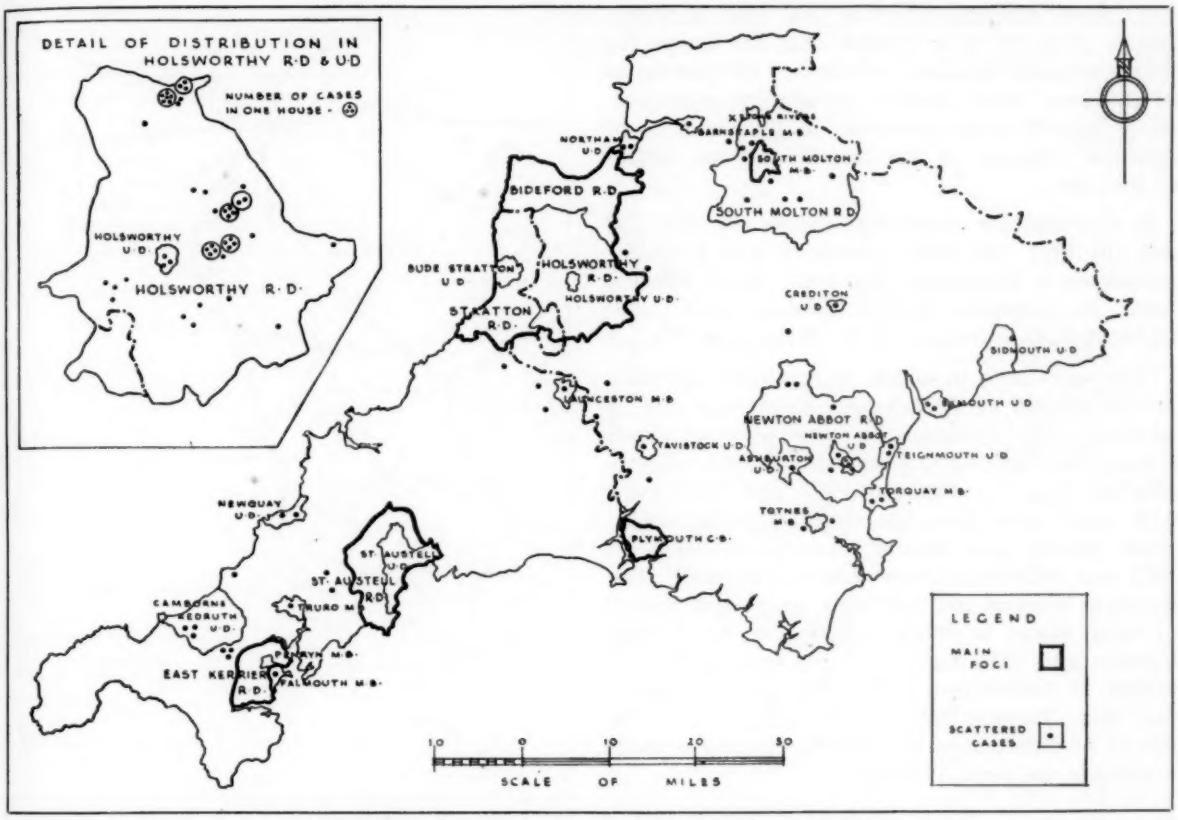

FIG. 3.—Distribution of poliomyelitis, 1911 (modified from Reece, 1912).

Outbreak in Plymouth.—In 1911, what is now the county borough of Plymouth was divided administratively into the county boroughs of Plymouth and Devonport and the urban district of E. Stonehouse with an aggregate population of about 207,000 as compared with the population of Plymouth of about 220,000 in 1951. Soltau (1911) said that he knew of 73 cases in the three towns and considered that this was far fewer than the actual number. He laid stress on the fact that the cases were scattered all over the three towns and that there seemed to be no relation between high prevalence and bad social conditions. If anything the better part of Plymouth suffered more severely and some of the poorest parts very little.

The outbreak in Plymouth began in July, later than in Holsworthy, and went on until October, in which month a few isolated cases occurred. That is all the epidemiological information we have about this outbreak, but it does suggest that incidence must have been considerably higher than it has ever been since.*

There were no cases in Exeter.

When the available information has been examined, the general impressions are:

(1) that it was more localized than epidemics have been in recent years,
(2) that the areas chiefly affected were rather those having little contact with the rest of England than the holiday resorts,
(3) that attack rates in some of the districts affected were quite as high as those which have since occurred, even in years like 1949 and 1950.

It is difficult to imagine how a distribution of cases such as that of 1911 could have resulted from immediate importation of infection by holiday-makers, particularly in view of the primitive communications which existed at that time in these two counties.

INTER-EPIDEMIC PERIOD, 1912–46.

(a) 1912–36.—After the epidemic of 1911 came a long period during which poliomyelitis never became sufficiently prevalent in the two counties to excite comment in the Annual Reports of the County Medical Officers. For a few years after the first world war, the figures of annual notifications for Cornwall and Plymouth were somewhat high but

* The highest number of cases reported in Plymouth in any year from 1912 to 1952 was 31 in 1950.

they never reached thirty in one year in either county (Fig. 1). It is possible that the figures for these years may have been affected by the prevalence of the then "new" disease encephalitis lethargica, which caused some confusion in the statistics of epidemic diseases of the central nervous system at the time.

In Cornwall the rather high figures for 1919 (24) and for 1921 (24) were associated with a similar prevalence in Plymouth. The areas chiefly affected were St. Germans R.D., Liskeard R.D., and Calstock R.D. (now part of St. Germans R.D.).

The year 1926, in which occurred the epidemic in the schools of Broadstairs, Kent, was one of relatively high incidence in many parts of Great Britain, but Devon and Cornwall were only slightly affected (Fig. 1). Between 1922 and 1936 there were never more than ten annual notifications in either county and usually there were fewer. In 1923 and 1929 there were none in Cornwall. The Registrar-General did not begin to publish figures of notifications in county districts in his Annual Review until 1931 and so it is not easy to find the details of distribution within the counties before that year. There is little of note to be found in the figures of distribution by districts of the few cases notified in the years 1931–36.

(b) 1937–46.—In 1937 there was a small localized epidemic in Devon. The outbreak began in a village called Drewsteignton which lies in the angle between the trunk road A.30 and the main road A.382 from Whiddon Down-Moretonhampstead-Newton Abbot-Torquay-Paignton. The village is just off both roads—about a mile S. of A.30 and 4 miles E. of A.382 (Fig. 4). There were six cases in Drewsteignton and a series of cases in places on either side of A.382—two at Moretonhampstead, two at Lustleigh, one at Manaton, three at Hennock, one at Widdecombe, one at Kingsteignton, and one at Denbury. Four cases were reported in Torquay, and two of these, in a preparatory school, were associated with a case in Hennock. At about the same time (September–October) as this group of cases, there were widely scattered cases in other parts of the county and for the whole year the Registrar General gives 33 notifications for the Administrative County, three for Plymouth, and two for Exeter. It should be remembered that 1938 was the year of epidemic prevalence in England and Wales as a whole and that this incident in Devon preceded that prevalence.

Neither Cornwall nor Devon seems to have been affected by the epidemic of 1938.

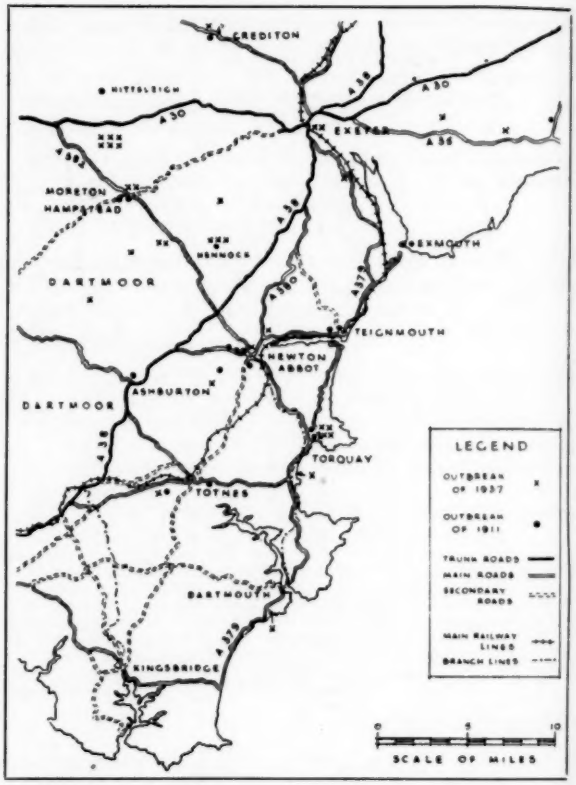

FIG. 4.—Distribution of poliomyelitis in the Newton Abbot and South Devon area, 1937. Figures for 1911 are also shown for comparison.

In the war years there were no incidents sufficiently important to arouse comment at a time when everyone was pre-occupied with other matters, but a study of the notification returns produces some small points of interest in the light of events which came after:

In Cornwall in 1941 there were nineteen notifications with three each in St. Austell U.D., St. Germans R.D., and Truro R.D., and two each in Liskeard U.D., and St. Austell R.D. In 1945 there were 29 notifications in the county with seven in Kerrier R.D., six in Camborne-Redruth U.D., and two each in Bodmin, Falmouth, St. Austell U.D., Camelford R.D., and W. Penwith R.D.

In Devon in 1944 there were 23 notifications, with five in Torquay, four in Ottery St. Mary, and two each in Paignton, Barnstaple R.D., and Plympton St. Mary R.D.

The experience of the inter-epidemic period may therefore be summarized as follows:

1919–21.—There is a little evidence of rather high prevalence in Plymouth and in the neighbouring rural districts of E. Cornwall.

1937.—There was a localized outbreak in Devon affecting the S.E. corner of Okehampton R.D., and Newton Abbot R.D. immediately to the S. of it.

1945.—There were 29 notifications in Cornwall, with seven in Kerrier R.D., and six in Camborne-Redruth U.D.

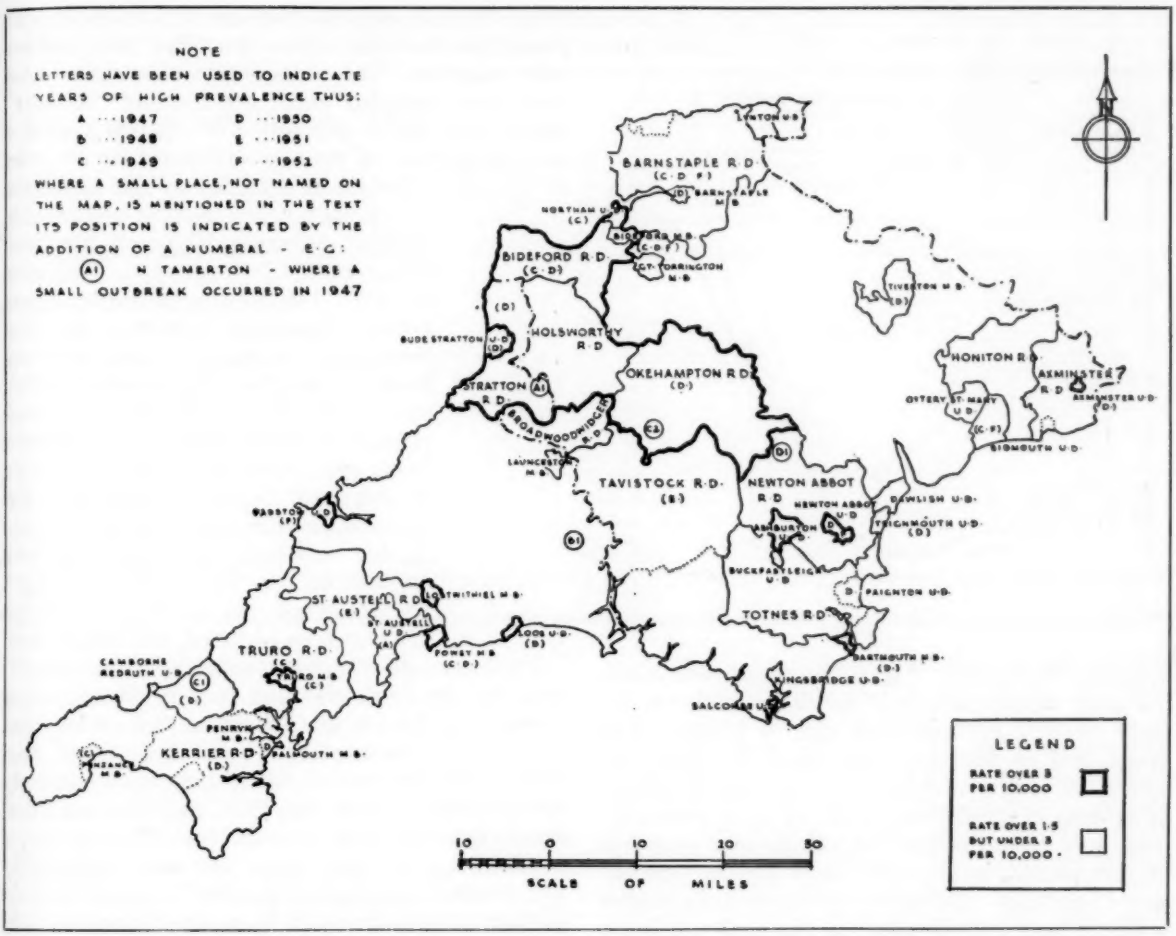
NOTE
LETTERS HAVE BEEN USED TO INDICATE YEARS OF HIGH PREVALENCE THUS:
A ···1947
B ···1948
C ···1949
D ···1950
E ···1951
F ···1952
WHERE A SMALL PLACE, NOT NAMED ON THE MAP, IS MENTIONED IN THE TEXT ITS POSITION IS INDICATED BY THE ADDITION OF A NUMERAL - E.G.:
(A1) N TAMERTON - WHERE A SMALL OUTBREAK OCCURRED IN 1947
BARNSTAPLE R.D.
BIDEFORD R.D.
HOLSWORTHY R.D.
STRATTON R.D.
OKEHAMPTON R.D.
TAVISTOCK R.D.
NEWTON ABBOT R.D.
TOTNES R.D.
ST AUSTELL R.D.
TRURO R.D.
KERRIER R.D.
HONITON R.D.
AXMINSTER R.D.
LEGEND
RATE OVER 3 PER 10,000
RATE OVER 1·5 BUT UNDER 3 PER 10,000
10 0 10 20 30
SCALE OF MILES

FIG. 5.—Distribution of poliomyelitis, 1947–52. Mean annual rates of notifications and localized epidemics.

RECENT EPIDEMIC PERIOD, 1947–52

In England and Wales as a whole the year 1947 was one of great change in the epidemiology of poliomyelitis. In Cornwall and Devon, Exeter and Plymouth, the change was not so marked as in other parts of the country, but there was a definite increase in notifications (Fig. 1). Although this part of England largely escaped the epidemic of 1947, both administrative counties experienced a high incidence in 1949–50 and in these years their rates were higher than the average for England and Wales (Table I).

The mean annual rates for individual county areas and the notifications in each year are shown in the detailed Tables of the Appendix. Populations taken from the preliminary census report are also shown, since some of the areas have such small populations that a few cases produce a high rate.

Fig. 5 is intended to summarize the information in the detailed Tables and to illustrate it in geographical terms.

Some of the urban districts, in Cornwall particularly, cover quite a large area and include various villages within their boundaries. For example Camborne-Redruth U.D. includes the village of Illogan where there was a sharp outbreak in 1949.

For the whole period, Cornwall A.C. had the highest rate and both Devon A.C. and Exeter C.B. had a higher rate than that for England and Wales, but Plymouth C.B. had a rate only half that of Cornwall and substantially less than that for the whole country. There was no consistent difference in the rates for urban and rural districts, for in Cornwall A.C. the urban districts had a higher rate (1·8 per 10,000) than the rural districts (1·2 per 10,000), whereas in Devon A.C. the rural districts had a slightly higher rate (1·5 per 10,000) than the urban districts (1·4 per 10,000).

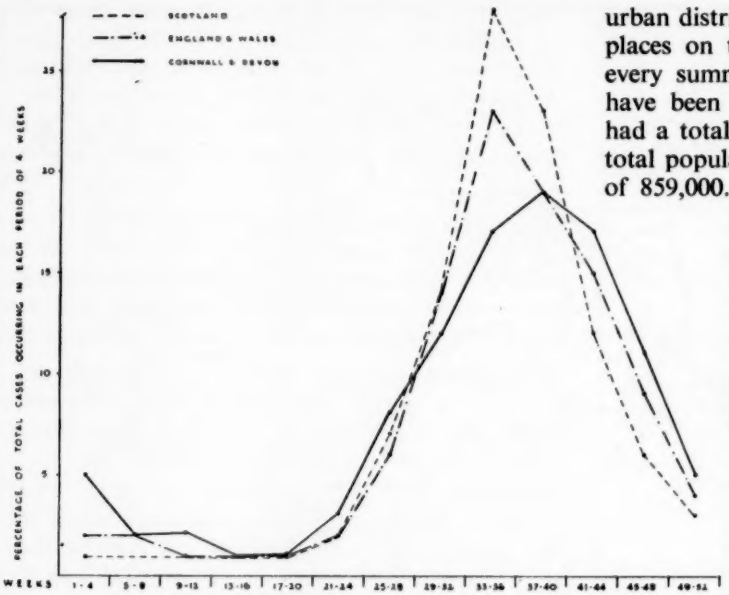

FIG. 6.—Uncorrected notifications of poliomyelitis in 4-weekly periods, 1947–51.

SEASONAL INCIDENCE.—Fig. 6 shows how the seasonal incidence of notifications in the two counties in the years 1947–51 differed from that in England and Wales and in Scotland. In Scotland, where the season of warm weather is comparatively short, 28 per cent. of notifications occurred in the 33rd–36th week period. In England only 23 per cent. occurred in this period, in Cornwall and Devon together only 17 per cent. The curve for the western counties is flatter than that for either of the others, and the poliomyelitis season begins a little earlier and lasts a little longer into the autumn and winter. This difference between North and South has been described in the U.S.A. (Serfling and Sherman, 1953).

IMPORTATION OF INFECTION.—If this is an important immediate factor in the genesis of epidemics, it would seem reasonable to expect that the ring of seaside resorts which encircle the two counties would tend to have high rates of incidence. This problem is examined in Table III. The Table is open to the objection that it takes account only of these resorts which are municipal boroughs or urban districts whereas there are very many smaller places on the coast which are filled with visitors every summer. The coastal urban districts which have been included under the heading "Resorts" had a total census population of 263,000 against a total population of the two administrative counties of 859,000. The places included in the aggregate of "Resorts" are Seaton, Sidmouth, Budleigh Salterton, Exmouth, Dawlish, Teignmouth, Torquay, Paignton, Brixham, Dartmouth, Salcombe, Looe, Fowey, Falmouth, Penzance, St. Ives, Newquay, Padstow, Bude-Stratton, Northam, Bideford, Barnstaple, Lynton, Ilfracombe. The notifications in these places have been taken from the Appendix Table and a rate calculated by dividing by the total census population. The remaining notifications have been divided by the remaining population to give the rates for the "Remainder of A.C.s". The combined rate for the two counties has also been included for comparison.

TABLE III

NOTIFICATION RATES PER 10,000 LIVING 1947–52

Year	1947	1948	1949	1950	1951	1952	Mean 47–52
"Resorts" ..	0·9	0·2	2·4	3·7	0·6	1·2	1·5
"Remainder of A.C.s" ..	1·1	0·6	2·3	3·0	1·2	0·8	1·5
A.C.s (Combined) ..	1·1	0·5	2·3	3·2	1·0	0·9	1·5

There is no difference between the mean (1947–52) rates for the "Resorts" and for the counties as a whole. The figures for individual years show that in 1947 the "Resorts" rate was a little lower than that for the rest and in the two years of relatively low prevalence, 1948 and 1951, the "Resorts" had definitely lower rates than the rest. The figures of notifications for these years are very small (only five notifications in the "Resorts" in 1948) and so perhaps not much should be made of them, but the difference is consistent in both years.

The figures for the individual resorts given in the Appendix Table show that the smaller ones (*e.g.* Fowey, Dartmouth) contributed far more notifications in proportion to their size than did the larger ones (*e.g.* Torquay, Paignton, Penzance). On the whole this evidence seems to be against the view that the principal resorts play any obvious part in the genesis of the epidemic.

One of us (E.R.H.) has examined the known instances of importations (*i.e.* persons developing obvious disease within a week of arrival) of the disease into Cornwall in the individual years 1947–52 and has summarized the information (Table IV, opposite).

It seems clear that the arrival of overt cases of poliomyelitis was not an important factor in Cornwall but this evidence of course tells us nothing about the effect of inapparent importation and for this we must turn to a more detailed consideration of events in the two counties in the individual years.

TABLE IV

CORNWALL: KNOWN IMPORTATIONS (1947–52)

Year	Total Cases	Importations	Remarks on Importations			
			No.	Town	District	Recurrence
1947	36	4	1	Portreath	Camborne-Redruth U.D.	—
			1	Marazion	W. Penwith R.D.	—
			2	St. Ives		—
1948	19	0	—	—	—	—
1949	11	4	2	St. Ives		one in a child of 3 1 month later but no connexion traced
			1	Megavissey	St. Austell R.D.	—
			1	Marazion	W. Penwith R.D.	—
1950	98	12	3	Looe		—
			1	Lanlivery	St. Austell R.D.	—
			1	Newquay		—
			1	Manaccan	Kerrier R.D.	—
			1	Padstow		—
			1	Redruth		
			1	St. Blazey	St. Austell U.D.	—
			3	Bude-Stratton		14 cases after 36-day interval
1951	36	3	1	Newquay		—
			2	Pentewan	St. Austell U.D.	—
1952	21	1	1	Falmouth		—

1947–48, Cornwall.—The first cases in Cornwall occurred in July, 1947, at North Tamerton, a remote village near Holsworthy (Fig. 5, A.1). This outbreak was remarkable for the high proportion of non-paralytic cases, and bears some resemblance to that at Stoke Rivers in 1911, described by Reece (1912), which has already been mentioned. At North Tamerton, however, there were three paralytic cases whereas at Stoke Rivers there had been none. The outbreak has been described by one of us in an earlier paper (Hargreaves, 1949). It is a curious fact that these first cases occurred so near to the main focus of the epidemic of 1911 (see Figs 2 and 5.)

The second outbreak appeared in the St. Austell area early in August. The first case (August 10), was that of a girl aged 7 living at Trenoweth Mill, a very rural spot some 5 miles from St. Austell. No other case had occurred within 30 miles. There was an interval of 34 days before the second case on September 12, of a woman aged 26 who was a direct contact of the previous case, in that the little girl was in her Sunday School class. The woman worked in St. Austell, and on October 22, 40 days later, two girls, both indirect contacts of hers living in St. Austell, developed poliomyelitis. A further six cases occurred in the town in the following 3 weeks, the actual dates of onset being:

October 30—8 days interval (one case)
November 6 (two cases)
November 10 (two cases)
November 12 (one case).

In two of the last cases, direct contact had occurred (7 days and 21 days previously); in the remaining four cases, indirect contact was probable.

The outbreak spread in a leisurely fashion eastwards along the main trunk road. In November, a case occurred at St. Blazey 3 miles east of St. Austell, and a month later two cases occurred at Lostwithiel, 7 miles east of St. Austell, and one case at East Taphouse, still further to the east.

Callington (Fig. 5, B.1) lies on the same trunk road, 31 miles east of St. Austell, and here at the turn of the year an explosive outbreak occurred. In all, there were six cases, five probably infected from the same source, since they developed paralysis between December 29, 1947, and January 2, 1948; the sixth, a secondary case, occurred on January 11, 1948, 8 days later.

1947–48, Devon.—In Devon there were no such definite groups of cases as there were in Cornwall, but the following districts stand out a little from the rest as having reported an unusually large number of cases:

URBAN DISTRICTS	Newton Abbot U.D.	4
	Torquay M.B.	4
RURAL DISTRICTS	Holsworthy R.D.	5
	Newton Abbot R.D.	4
	St. Thomas R.D.	6
	Tavistock R.D.	5

Torquay has a population of 53,216, so perhaps four notifications are not so very remarkable. St. Thomas R.D. is also a populous district, since

it includes the suburbs of Exeter, and Exeter had a slightly higher attack rate than Devon A.C. in 1947 (Table I). Holsworthy R.D., the main focus of the epidemic of 1911, reported five cases in a population of only 6,196.

Allen-Price (1952) described the distribution of cases in West Devon (Tavistock U.D. and R.D., Okehampton U.D. and R.D., and Broadwoodwidger R.D.) over the period 1937–51; writing of 1947 he says:

Ten cases occurred in nine parishes with many miles and poor communications separating them. Once again there was no spread from the individual cases, with the exception of two cases in the same family at Peter Tavy, a small village some 4 miles from Tavistock.

In 1948 there was virtually nothing to report in Devon, and Newton Abbot R.D. was the only district which reported as many as three cases (see Appendix Table).

1949–50, Cornwall.—This was the worst period that Cornwall has yet experienced. 111 cases were confirmed in 1949, giving an incidence of 3·5 per 10,000. In 1950 there were 98 cases. Four interesting localized outbreaks occurred at Illogan, Camborne-Redruth, Fowey, and Truro (see Fig. 5; Illogan C.1).

(*i*) Illogan is a village some 3 miles from Camborne. Three children were attacked on June 21 and all had paralysis. Two were pupils at the Illogan Infants School, and the third, a child of three, had an elder sister there. After an interval of some 7–10 days, five secondary cases occurred all intimately connected with the school, four being pupils, and direct contacts of the initial cases, and the fifth a child of 2½, who had a brother at the school. The source of the original cases was not discovered but subsequent spread was probably by personal contact at the school.

(*ii*) Outbreaks occurred in Camborne and Redruth almost simultaneously, and in both some connection with Illogan cases was easily demonstrated. At Redruth the original three cases had all been to Illogan by bus 10 days before the onset. At Camborne, the first patient was a cousin of an Illogan patient, and his mother, the patient's aunt, in spite of warnings, had visited him on two occasions, the last being 8 days before the onset of paralysis. The outbreaks thus begun, resulted in a further twelve cases.

(*iii*) Two serious outbreaks occurred in Fowey in successive years. The town (population 2,300) had in 1949 eight cases of poliomyelitis (incidence 35 per 10,000), and in 1950 a further fifteen cases (incidence 64 per 10,000). The outbreak of 1949 came to the surface in August when five children developed paralytic poliomyelitis within 48 hrs of one another. Secondary cases numbered only three, all being personal contacts with the original cases. The origin of the primary cases was not found. Although the outbreak at Fowey did not show itself until August, three visitors who had been staying in the town in July developed paralytic poliomyelitis within 5 days of returning to their several homes in Cambridge, London, and Scunthorpe. In 1950 a more severe outbreak occurred; the first case developed symptoms on September 2, and between this date and the first week of November fourteen more cases occurred in the town and three in the immediate neighbourhood. All but two patients were of school age or under, and were directly or indirectly associated with the local school.

(*iv*) There were no cases of poliomyelitis in Truro (population 12,851) during 1947. In the autumn of 1948, three cases, two of which proved fatal, occurred amongst boarders at a boys' school in the town. In 1949 the city was heavily attacked; two paralytic cases were reported on September 11, and in the subsequent 10 days twelve more cases were admitted to the County Isolation Hospital. In all, nineteen cases occurred in the city and seven secondary cases in the surrounding villages. Paralysis was severe, particularly in early cases, three of the first five being fatal. The outbreak in Truro City presents certain interesting features:

(*a*) the high age group attacked—52 per cent. of the patients were over 15 years of age,

(*b*) no history of direct or indirect contact with previous cases could be obtained.

Bude-Stratton U.D. and Stratton R.D. (combined population 11,352) include the small towns of Bude, Stratton, and Marhamchurch, forming a triangle with sides approximately 1½ miles long (population 5,230), and the surrounding coast and countryside (population 6,122). In 1947 there was the outbreak at North Tamerton already mentioned and in subsequent years, the incidence in this urban district and the surrounding rural areas was heavy. In 1949 there were five cases, in 1950 seventeen, in 1951 two, in 1952 two.

The first case was notified on September 29, 1949, when a boy aged 7½ was admitted to Exeter with paralysis of both lower limbs. 12 days later, a father and son, both contacts of the first patient, contracted the disease, and the father subsequently died. Another contact became infected on November 16, 1949. The fifth case to be notified was a school child who at routine inspection was found to have a weak arm. The date of onset was uncertain.

In 1950, the first three cases were imported: two were soldiers from a nearby territorial camp, and the third was a girl in a visiting circus. All developed the disease within 4 days of arrival in the Bude area. The subsequent fourteen cases which occurred in October, 36 days after the last of the imported cases, and during the first 3 weeks in November, were all local residents. The first, a boy of 8½, developed paralytic poliomyelitis and died at Plymouth I.D. Hospital the following day. The case is of special interest as this is the same child who in

1949 was admitted to hospital with paralysis of the two lower limbs. His baby brother, 9 months old, developed a mild attack 6 days later, but the remaining three children in the house were unaffected.

The spacing of cases again suggests contact spread and the possibility was borne out by definite history of contact in five of the first six cases.

The 321 Cornish cases in the years 1947–52* included twelve instances of paralytic poliomyelitis occurring in two members of the same household, and two instances of three cases occurring in one household. If non-paralytic cases diagnosed at home on clinical symptoms are included, the number of multiple infections is much greater.

Of these multiple cases, in six instances the two cases developed paralysis within 24 hrs. of each other, in eight instances the interval was between 4 and 9 days, in one case a 20-day interval occurred, and in one the third child was not affected until the following year.

Three of these multiple cases were of special interest:

(*i*) In 1949 a Dutchman, his English wife, and three children aged 5½, 4½, and 3 years, were occupying an overcrowded condemned three-roomed cottage in one of the older parts of Fowey. Early in August, the youngest child developed poliomyelitis; the two older children, who had been in intimate contact with the sick child developed symptoms suggestive of abortive attacks of poliomyelitis, but were not taken to hospital. During the spring of 1950, the family moved to a new Council house high up on the outskirts of the town. In October, 1950, the two older children developed poliomyelitis, the interval between onset being 5 days. The old house vacated by this family was not pulled down, but was taken by a young married couple with three children. The wife, a woman of 30, died of poliomyelitis in November, 1950.

(*ii*) A Truro man aged 27 was taken ill on September 9, 1949, and died 2 days later of respiratory paralysis. There were three other siblings in the family, all grown up, and they were all at home at the time of his illness. A brother, a naval officer stationed in Plymouth, was in Truro on a week's leave and assisted in nursing him. He returned to Plymouth and went down with a mild attack of poliomyelitis on September 30. An unmarried sister who helped in the clerical work of the family business and had been in close contact with the brother who had died, was taken ill on September 25, and developed paralysis of all four limbs with diaphragmatic involvement on September 29. The remaining brother, also in the Navy, was unaffected. The history of this family illustrates how infective poliomyelitis virus can sometimes be.

(*iii*) A woman was delivered of her second child on September 1; apart from slight pyrexia on September 14, the puerperium was uneventful. She was up in the house from September 15–19, but did not feel very fit. On September 20 she was feverish with nausea and headache; next day she was no better and as the baby was also off colour and feverish it was decided to begin artificial feeding. The mother remained in bed until September 28, when on getting up she first noticed a weakness in her left leg, which proved to be poliomyelitis. Artificial feeding was easily established, but on September 25 and 26 the baby had four convulsions and subsequently developed some weakness in the muscles of the shoulder girdle.

Recurrent infection in the same house leads to the idea of the possibility of persistent carrier state; the following incidents are suggestive though they may be due to chance.

(*i*) *Padstow.*—In October, 1949, a girl of 7 developed an illness diagnosed as non-paralytic poliomyelitis, but not notified as such. In August, 1950, a child living in the next house but one developed poliomyelitis. Both families moved, the former to Tintagel (but keeping a flat at their Padstow house), and the latter out of the county. In January, 1952, a boy of 12 who had come to live in the second house developed poliomyelitis. He was also a friend of the first original case, and met her on her frequent visits to the Padstow flat. Almost at the same time, a girl living in Tintagel but at boarding school in mid-Wales, who was an acquaintance of the first patient, developed poliomyelitis 4 days after returning to school. In none of the incidents cited, was poliomyelitis prevalent in the area.

(*ii*) *St. Ives.*—In January, 1950, a married woman developed paralytic poliomyelitis whilst in Malta; she returned to England 2 years later and stayed in St. Ives at the house of her married sister. Within a month the sister was admitted to hospital with paralytic poliomyelitis. This was the only case of poliomyelitis in St. Ives at the time.

(*iii*) *Fowey.*—The history of the Dutchman's house already cited in the notes on multiple cases.

1949–50, Devon.—In 1949 incidence in Devon was slightly higher than that in the country as a whole, but considerably lower than that in Cornwall (Table I). In June, 1949, there was an explosive outbreak in the village of Bridestowe, Okehampton R.D. (Fig. 5, C.2), which occurred at almost the same time as that at Illogan (Fig. 5, C.1). This event has been described in some detail by Allen-Price (1952), according to whom there were thirteen cases between June 17, and July 12. Evidently two of these were not notified as the official return for Okehampton R.D. for 1949 gives eleven cases (see Appendix Table). The village of Bridestowe, population about 550, lies on the trunk road A.30 and on the N.W. edge of Dartmoor. The trunk road carries a very large volume of holiday traffic, but

the outbreak occurred very early, at a time when the seasonal rise in notifications in the country as a whole had only just begun. The attack rate was very high and Allen-Price states that 18 per cent. of the child population was affected. This was the most important localized outbreak in Devon in 1949, but the districts of the N.W. round Bideford and Barnstaple and the town of Sidmouth had a high incidence.

The rate in 1950 (3·5 per 10,000) was the highest recorded for the Administrative County, and the County Borough of Exeter also had its highest incidence (3·4 per 10,000).

The following summary of the distribution of cases is based on that given in the report of the County Medical Officer of Health for 1950:

> Incidence was not unusual until August when there was a sharp rise in the weekly notifications. At first the districts chiefly affected were Newton Abbot U.D. and Teignmouth U.D., and there were three scattered cases in Newton Abbot R.D., and one or two in fourteen other districts. In September there was a localized outbreak of thirteen cases in Dartmouth U.D. (population 5,842). In October, there was another localized outbreak of eleven cases at Moretonhampstead, a small town (population about 1,500) on Dartmoor (Fig. 5, D1). Once again the districts of the N.W. had a relatively large number of cases but no localized outbreak.

1951–52, Cornwall.—Incidence declined in these years to 1·1 per 10,000 in 1951, and 0·6 in 1952. The main features were the persistence of cases in the Camborne, Redruth, and St. Austell areas, with a minor outbreak at St. Mawgan (St. Austell R.D.) of six cases during March–April, 1951, and the occurrence of four cases at Padstow in 1952.

1951–52, Devon.—Incidence declined to 1·0 per 10,000 in 1951, but rose slightly to 1·2 in 1952. Tavistock R.D. had eight notifications in 1951, but these were mostly scattered round Tavistock itself. In 1952, Newton Abbot U.D., the districts of the N.W. corner, and Sidmouth had an excessive number of notifications.

Discussion

The chief value of a study such as this may be to suggest ways in which the new tools of epidemiological investigation now becoming available may best be used. It would, for example, be interesting to know whether the outbreak in the small town of Fowey in 1950 was caused by a different strain of virus from that of 1949 or whether the two outbreaks were really one, with an intervening period of quiescence. One might even go further and speculate whether the Fowey outbreak of 1949 was associated with the prevalence in the neighbouring town of St. Austell in 1947.

The most suggestive facts which emerge from the survey are first the recurrence of the disease in certain areas, and secondly the fact that the big resorts do not seem to have played an obvious part in the genesis of epidemics in the two counties. There are a few areas which have enjoyed a remarkable freedom from the disease over the years. In Cornwall, St. Just U.D., and the Scilly Isles, and in Devon, South Molton U.D., have not had a notified case at least since 1931. These places have very different social characteristics. The Scilly Isles are not very easily accessible from the mainland, but a fair number of holiday makers do go there in the spring and summer. St. Just is a small mining town where social conditions are poor, and South Molton is a market town on the main trunk road through North Devon (A.361). South Molton U.D. was a secondary focus in the epidemic of 1911 and is the most striking example of non-recurrence to be set against the examples of recurrence illustrated in Figs 2 and 5. It would be interesting to know whether the herd immunity in these places is different from that in areas of high incidence.

In the detailed notes on recent events in Cornwall, attention has been drawn to certain occasions when it has seemed that infection has persisted over a long period either in a family, or, in one instance, in a house. These have been mentioned because other observers of the disease may also have come across curious happenings of this kind. They seem quite inexplicable on the generally accepted theories of duration of infectivity and mode of spread, and they may be due to chance.

It is very difficult to formulate any epidemiological theory which will fit all the facts of distribution, though it is easy enough to select some of the facts and devise theories to suit them. For example, one might argue that the distribution of 1911 in the Administrative Counties suggested a low herd immunity in the rural population, but it is difficult to reconcile this argument with the high incidence in Plymouth. Again, one might argue that the relative freedom of these two western counties from the epidemic of 1947 suggests that a new virus was imported into eastern England in 1947 and took some time to show its effects in the West. It is, however, difficult to reconcile this argument with the distribution of those cases which did occur in Devon and Cornwall in 1947.

Several examples have been given which make it hard to believe that a localized epidemic generally

ends because of the exhaustion of susceptibles. Fowey is the chief of these but Newton Abbot and the districts of the North coast are lesser ones.

As has been said, there is little evidence that epidemics start in the big resorts. It is, however, possible that there may be a silent building-up process in the big resort whilst the overt outbreak occurs much later in a smaller place.

In reviewing the facts of distribution in a general way, we are left with a general impression that a silent building-up period may be very important, and that the overt outbreak is the culmination of a process which may have been going on for some time, possibly for a year or two. This idea must remain pure speculation as long as the only indication we have of the presence of the virus is the occurrence of frank cases, but when it becomes possible to look for the virus in apparently healthy populations and to investigate the antibody content of blood samples it may be possible to learn more about the genesis of outbreaks.

SUMMARY

(1) A study has been made of the distribution of notifications of poliomyelitis in Cornwall and Devon from 1911 to 1952.

(2) No direct evidence has been found of importation of infection from other parts of England into these two holiday counties.

(3) There is a suggestion that outbreaks tend to recur in the same, rather ill-defined areas, sometimes at intervals of many years, but there is no obvious social characteristic common to these areas.

Our thanks are due to the Medical Officers of Health of Cornwall and Devon, Exeter and Plymouth and to members of their staffs; to many District Medical Officers of Health and to Miss V. R. Harrison, A.R.I.B.A., who prepared the maps for us.

REFERENCES

Allen-Price, E. D. (1952). *J. roy. sanit. Inst.*, **72**, 443.

Benjamin, B., and Logan, W. P. B. (1953). *British Journal of Preventive and Social Medicine*, **7**, 131.

Cornwall County Council Annual Reports of the County Medical Officer of Health, 1911–51.

Devon County Council Annual Reports, County Medical Officer, 1911–51.

Gregor, A., and Hopper, L. B. (1911). *Brit. med. J.*, **2**, 1154.

Hargreaves, E. R. (1949). *Lancet*, **1**, 969.

Reece, R. J. (1912). "Reports to Local Government Board on Public Health and Medical Subjects", N.S. No. 61, p. 10. H.M.S.O., London.

Registrar-General Statistical Review of England and Wales, Part 1. Tables (Medical), 1921–51.

Serfling, R. E., and Sherman, I. L. (1953). *Publ. Hlth Rep., Wash.*, **68**, 453.

Soltau, A. B. (1911). *Brit. med. J.*, **2**, 1151.

APPENDIX TABLE

DEVON

Area	Population Census 1951	Mean Annual Rate per 10,000 1947–52	Number of Notifications in each Year						Total	Fig. 5
			1947	1948	1949	1950	1951	1952	1947–52	
COUNTY BOROUGHS										
Exeter	75,479	1·4	10	6	6	25	4	10	61	
Plymouth	208,985	0·8	8	3	20	31	26	9	97	
ADMINISTRATIVE COUNTY— URBAN DISTRICTS										
Ashburton	2,704	3·7	2	1	—	3	—	—	6	1
Axminster	2,673	4·4	—	—	—	7	—	—	7	1
Barnstaple	16,302	2·2	1	—	2	11	3	4	21	2
Bideford	10,100	3·1	—	—	8	6	—	5	19	1
Brixham	8,761	1·0	—	—	1	3	1	—	5	
Buckfastleigh	2,592	1·9	—	2	—	1	—	—	3	2
Budleigh	3,953	—	—	—	—	—	—	—	—	
Crediton	3,992	0·8	—	2	—	—	—	—	2	
Dartmouth	5,842	4·3	—	—	1	13	1	—	15	1
Dawlish	7,512	1·6	3	1	1	2	—	—	7	2

N.B.—Fig. 5: 1, Heavy outline; 2, Light outline

APPENDIX TABLE—*cont.*

DEVON—*cont.*

Area	Population Census 1951	Mean Annual Rate per 10,000 1947–52	Number of Notifications in each Year						Total	Fig. 5
			1947	1948	1949	1950	1951	1952	1947–52	
ADMINISTRATIVE COUNTY— URBAN DISTRICTS										
Exmouth	17,232	0·2	—	—	1	—	—	1	2	
G. Torrington	2,872	1·7	—	—	—	—	3	—	3	2
Holsworthy	1,550	—	—	—	—	—	—	—	—	
Honiton	4,614	0·7	—	—	—	2	—	—	2	
Ilfracombe	9,218	0·4	—	—	—	1	—	1	2	
Kingsbridge	3,153	2·6	—	1	—	2	2	—	5	2
Lynton	2,123	1·6	—	—	—	—	2	—	2	2
Newton Abbot	16,393	3·5	4	1	2	14	2	11	34	1
Northam	6,470	3·1	1	—	9	1	—	1	12	1
Okehampton	3,897	0·4	—	—	1	—	—	—	1	
Ottery	4,015	1·7	1	—	1	—	2	—	4	2
Paignton	25,369	1·1	3	—	4	6	3	—	16	
Salcombe	2,576	3·2	—	—	—	1	2	2	5	1
Seaton	2,903	0·6	1	—	—	—	—	—	1	
Sidmouth	10,043	2·4	1	—	7	1	—	6	15	2
South Molton	3,125	—	—	—	—	—	—	—	—	
Tavistock	5,889	0·9	1	—	1	—	1	—	3	
Teignmouth	10,589	1·7	2	—	—	9	—	—	11	2
Tiverton	10,869	1·7	—	1	3	7	—	—	11	2
Torquay	53,216	0·4	4	2	—	4	1	1	12	
Totnes	5,534	0·3	—	—	1	—	—	—	1	
RURAL DISTRICTS										
Axminster	11,516	1·2	—	1	—	5	—	2	8	
Barnstaple	23,582	2·1	5	—	8	6	1	10	30	2
Bideford	5,544	3·9	—	—	6	6	—	1	13	1
Broadwoodwidger	2,028	1·6	1	—	—	—	1	—	2	2
Crediton	10,041	0·8	—	1	1	1	2	—	5	
Holsworthy	6,196	3·0	5	—	—	3	3	—	11	1
Honiton	7,131	1·6	—	1	1	1	2	2	7	2
Kingsbridge	11,804	1·1	1	1	1	4	—	1	8	
Newton Abbot	25,795	2·5	4	3	1	28	1	2	39	2
Okehampton	12,621	3·2	1	—	11	9	2	1	24	1
Plympton St. Mary	31,790	0·9	—	—	5	7	2	3	17	
St. Thomas	31,919	0·9	6	1	4	6	—	1	18	
South Molton	9,087	1·1	—	—	—	4	2	—	6	
Tavistock	16,300	1·3	5	—	—	—	8	—	13	
Tiverton	20,714	1·0	2	1	4	3	1	1	12	
Torrington	7,387	0·2	—	—	1	—	—	—	1	
Totnes	13,923	1·6	1	2	2	3	2	3	13	
TOTAL—DEVON A.C.	513,819	1·5	55	22	88	180	50	59	454	

N.B.—Fig. 5: 1, Heavy outline; 2, Light outline

APPENDIX TABLE—*cont.*

CORNWALL

Area	Population Census 1951	Mean Annual Rate per 10,000 1947–52	Number of Notifications in each Year						Total	Fig. 5
			1947	1948	1949	1950	1951	1952	1947–52	
URBAN DISTRICTS										
Bodmin	6,058	—	—	—	—	—	—	—	—	
Bude-Stratton	5,230	2·9	—	—	2	7	—	—	9	2
Camborne-Redruth ..	35,829	1·8	1	1	26	6	5	—	39	2
Flamouth	17,036	1·4	1	—	3	8	1	1	14	
Fowey	2,344	17·1	—	—	8	15	1	—	24	1
Helston	5,545	0·6	1	—	—	1	—	—	2	
Launceston	4,467	2·2	1	—	2	3	—	—	6	2
Liskeard	4,391	1·1	—	—	1	—	1	1	3	
Looe	3,801	3·1	2	—	—	4	1	—	7	1
Lostwithiel	2,165	3·9	4	—	1	—	—	—	5	1
Newquay	9,928	1·5	3	—	3	1	1	1	9	
Padstow	2,852	3·5	—	—	—	2	—	4	6	1
Penryn	4,088	2·5	—	—	1	4	1	—	6	2
Penzance	20,648	1·3	—	2	9	2	—	3	16	
St. Austell	23,634	1·6	9	2	4	3	3	1	22	2
St. Ives	9,037	1·3	2	—	3	1	—	1	7	
St. Just	4,122	—	—	—	—	—	—	—	—	
Saltash	7,924	1·1	—	1	—	2	2	—	5	
Torpoint	5,852	0·3	—	—	—	1	—	—	1	
Truro City	12,851	3·4	—	3	19	1	1	2	26	1
RURAL DISTRICTS										
Camelford	7,577	0·4	—	—	1	1	—	—	2	
Kerrier	21,807	0·8	—	—	1	8	2	—	11	
Launceston	6,491	0·8	—	—	1	1	1	—	3	
Liskeard	14,312	0·9	3	1	1	3	—	—	8	
St. Austell	20,833	1·8	2	3	5	4	7	1	22	2
St. Germans	16,845	1·0	1	4	—	4	1	—	10	
Stratton	6,122	4·9	2	—	3	10	1	2	18	1
Truro	27,296	1·1	1	—	12	1	2	2	18	
Wadebridge	16,146	0·8	—	1	1	2	3	1	8	
West Penwith	18,216	1·3	3	1	4	3	2	1	14	
Scilly Isles	2,165	—	—	—	—	—	—	—	—	
TOTAL—CORNWALL A.C. ..	345,612	1·6	36	19	111	98	36	21	321	

N.B.—Fig. 5: 1, Heavy outline; 2, Light outline

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GASTRO-ENTERITIS IN INFANCY

FROM

THE PUBLIC HEALTH DEPARTMENT, LONDON COUNTY COUNCIL

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This paper describes a study of the epidemiology and the aetiology of infantile enteritis in two of the nine divisions into which the County of London is divided for day-to-day administration. The two divisions comprised the metropolitan boroughs of Chelsea, Fulham, Hammersmith, Kensington, Hampstead, Paddington, St. Marylebone, St. Pancras, and Westminster, with a total population of 994,000 at the 1951 census, and a total of 15,409 live births in 1950. The investigation began in July, 1949, and was continued until November, 1950. For the purpose of this study, enteritis was defined as diarrhoea and vomiting occurring in children under the age of 2 years, of such severity as to require admission to hospital (or to cause death before removal could be effected).

Enteritis is not a generally notifiable disease in London and it was necessary, therefore, to seek the co-operation of hospitals in a voluntary reporting system. This co-operation was freely forthcoming, and all fever hospitals, children's hospitals, and other hospitals likely to admit cases of diarrhoea in infants under 2 years of age agreed to notify the county medical officer of health of all such admissions.

Method of Inquiry

All these cases were registered in order to provide a general picture of the incidence of enteritis admissions. Those infants whose homes were in the survey area were the subject of domiciliary inquiry by health visitors for the appropriate districts. A whole-time health visitor was seconded for special duties in connection with the cases admitted to the enteritis unit at St. Ann's Hospital. A uniform questionnaire suitable for statistical treatment was designed, and the health visitors sought information on feeding and food preparation, size of family, occupation of principal wage-earner, housing and sanitation, previous medical history of the family, and possible sources of contact with infection.

When the domiciliary inquiries had been completed, the hospital was asked for an assessment of the severity of the illness, indications of parenteral infection on admission, information on bacteriological findings, and, when applicable, results of *post-mortem* examination.

Findings

Incidence.—Table I shows the trend of admissions during the year in the two selected divisions and in the County of London as a whole.

Table I

INCIDENCE OF REPORTED ENTERITIS ADMISSIONS IN 4-WEEK PERIODS THROUGHOUT THE YEAR ENDED JUNE 30, 1950

Period (wks)	Division 1	Division 2	London County
1–4	11	13	102
5–8	13	20	77
9–12	19	20	108
13–16	12	24	87
17–20	16	21	104
21–24	24	26	106
25–28	25	16	88
29–32	11	15	60
33–36	7	14	49
37–40	17	14	66
41–44	6	14	41
45–48	5	10	46
49–52	19	13	59
Total ..	185	220	993

Heavy concentrations of cases in infants admitted from families living in communal accommodation (rest centres, etc.) or from residential nurseries were observed. There was no evidence of any pronounced seasonal fluctuation. The falling off in reported cases in 1950 was part of a general decline in incidence of enteritis.

Sample.—The 360 cases reviewed included seventeen which were re-admitted to hospital after an initial period of treatment and which were only counted once each in respect of their total periods of treatment. Their inclusion depended to some extent on the reporting zeal of the hospitals, and 220 came from the enteritis unit of St. Ann's Hospital; but in so far as all reported patients resident in the specified

TABLE II

DISTRIBUTION BY AGE, FEEDING, SEX, AND BIRTH WEIGHT

Sex	Age (mths)	Feeding			Total	
		Wholly Breast-Fed	Partially or Wholly Bottle-Fed	Other	No.	Per cent.
Male	0–2	1	65	—	66	29·5
	3–5	4	50	—	54	24·2
	6–8	4	22	—	26	11·7
	9–11	4	27	4	35	15·7
	12–17	—	10	14	24	10·8
	18–23	—	1	17	18	8·1
	Not known	—	2	7	9	—
	Total ..	13	177	42	232	100·0
Female	0–2	5	24	—	29	23·4
	3–5	1	35	—	36	29·0
	6–8	2	23	—	25	20·2
	9–11	—	10	4	14	11·3
	12–17	—	4	9	13	10·5
	18–23	—	1	6	7	5·6
	Not known	1	3	—	4	—
	Total ..	9	100	19	128	100·0
Both		22	277	61	360	—
Percentage		6·1	77·0	16·9	100·0	—
Birth Weight (lb.)	2½	—	4	—	4	1·3
	3½	—	9	1	10	3·1
	4½	—	23	2	25	7·9
	5½	3	46	10	59	18·5
	6½	4	85	20	109	34·3
	7½	6	60	9	75	23·6
	8½	2	23	5	30	9·4
	9½ and over	1	4	1	6	1·9
	Not known	6	23	13	42	—
	Total	22	277	61	360	100·0
	Mean Weight	7·63	6·93	7·10	—	—
	S.E. of Mean	0·28	0·09	0·17	—	—

area were included it would seem reasonable to consider them as representative of hospital cases generally.

SEX AND AGE INCIDENCE (Table II).—There were nearly twice as many boys as girls, from which it would seem that in this disease, as in many others, there is a higher susceptibility in male than in female infants. The normal sex ratio of male to female infants under two years is 1·05 : 1 (about half of both boys and girls were under 6 months of age).

In the cases admitted to St. Ann's Hospital from their own homes, a graph of the age at onset of illness showed a marked peak at 1–2 months, a gradual decline in the following months, and a second peak between 11 and 12 months. The cases admitted from institutions showed an even incidence from 1 to 5 months of age; in most cases the illness began soon after admission to the institution.

BIRTH WEIGHT AND FEEDING (Table II).—As might be expected, very few (only 22, 6·1 per cent. *i.e.*) of the infants were completely breast-fed, the majority being bottle-fed or on mixed feeding.

Of the 22 breast-fed infants, thirteen had higher than average risks of exposure to infection (three came from rest centres, three had been hospital in-patients and one had been an out-patient in the 14 days preceding onset, two came from residential nurseries, one from a day nursery, and three were attending infant welfare centres); of these thirteen, eight had had a known contact with diarrhoea and vomiting within 14 days of onset. Of the remaining nine, four were known to have been in recent contact with enteritis. The breast-fed babies were not from especially poor families; of the fourteen for whom details were available, two were from Social Class II (as defined by the Registrar-General), eight from Class III, two from Class IV, and two from Class V (see also Table IV).

In the general population from which the sample was drawn, 76 per cent. of babies were breast-fed at 14 days and 31 per cent. were wholly breast-fed over the first 6 months of their lives (Douglas, 1950). Incidence of enteritis as defined was, therefore,

very much less in breast-fed than in bottle-fed infants, and so large a difference can hardly be accounted for by differential association with adverse socio-economic conditions, even though this factor is possibly related to the incidence of breast-feeding; nor is it wholly explained by the fact that many of the bottle-fed babies are nourished in that manner because they are prematurely born and are of low birth-weight. Even among the babies of normal birth-weight the breast-fed babies largely escaped disease of the character studied. The importance of breast-feeding emerges before that of any other preventive factor.

SEVERITY OF ILLNESS.—On discharge the cases were classified by the hospital physicians as follows (Table III):

About half the cases were mild or entirely without signs of diarrhoea during stay in hospital. Only one-fifth of all cases admitted to hospital were so severely ill with diarrhoea and vomiting as to require parenteral therapy. There were fifteen deaths, 4·6 per cent. of those with diarrhoea.

TABLE III
DEGREES OF SEVERITY OF ILLNESS

Symptoms	No.	Per cent.
No sign of diarrhoea	37	10·3
Mild	146	40·6
Moderate	106	29·4
Severe (requiring parenteral therapy)	71	19·7
Total	360	100·0

SOCIO-ECONOMIC BACKGROUND.—Sixty infants (16·2 per cent.) were illegitimate. This incidence of illegitimacy may be compared with the incidence of 9·6 per cent. in the general population of that area of London in which the study took place. The social class distribution of the cases may be compared with that of the general population of the county as measured in the 1951 census (1 per cent. sample) (Table IV).

It will be seen that the proportion of infants coming from Social Classes IV and V was higher (45·1 per cent.) than from the general population (29·0 per cent.). The proportion coming from Social Classes I and II was abnormally low (5·2 per cent., compared with a county average of 19·3 per cent.). Taken in conjunction with the higher incidence of illegitimacy, these differences, which are statistically significant, suggest that the incidence of enteritis is higher in the lower social classes. Some of the reasons for this differential will emerge later.

SIZE OF HOUSEHOLD.—The average size of household in each group (excluding cases in communal accommodation or residential nurseries) is shown in Table V (opposite), with comparative figures for the whole county as indicated by the 1951 census, and for the study area as obtained from health visitors' records.

The figures for the county as a whole are not strictly comparable; it is possible to refer only to households with at least one child under age 5, and for these the average size was 4·2. The average size of the households studied was 5·3 and it appears therefore, that the infants admitted to hospital were drawn from larger than average households in the county. In interpreting this difference, which is statistically significant and consistent in all sub-groups of disease severity, three independent influences must be borne in mind:

(1) Difficult social conditions are taken into consideration in allocating hospital beds, and it may be that, even if the incidence of enteritis were equal in all sizes of household, cases in the larger households would be more frequently taken into hospital care because of difficulties in home nursing.

TABLE IV
SOCIAL CLASS

Social Class*	Principal Wage Earner in Families Studied										Heads of Private Households (1951 Census, London A.C.)	
	Total Cases		No sign of Diarrhoea		Enteritis							
					Mild		Moderate		Severe			
	No.	Per cent.	No.	Per cent.	No.	Per cent.	No.	Per cent.	No.	Per cent.	No.	Per cent.
I	2	0·7	—	—	1	0·9	1	1·1	—	—	352	3·7
II	13	4·5	1	3·8	3	2·6	7	8·0	2	3·3	1,496	15·6
III	143	49·7	18	69·3	56	48·7	42	48·3	27	45·0	4,949	51·7
IV	77	26·7	7	26·9	23	20·0	25	28·8	22	36·7	1,268	13·3
V	53	18·4	—	—	32	27·8	12	13·8	9	15·0	1,503	15·7
Not known	72	—	11	—	31	—	19	—	11	—	1,576	—
Total ..	360	100·0	37	100·0	146	100·0	106	100·0	71	100·0	11,144	100·0

*As defined by Registrar-General (1951). "Classification of Occupations, 1950". H.M.S.O., London.

TABLE V
SIZE OF HOUSEHOLD

Average Number in Family		Total	No Sign of Diarrhoea	Enteritis			London A.C. 1951 Census*	L.C.C. Divisions 1 and 2 Health Visitors' Survey 1951*
				Mild	Moderate	Severe		
Adults		2·49	2·45	2·54	2·34	2·59		
Children	Under Age 2	1·45	1·24	1·43	1·50	1·52		
	Age 2 or Over	1·35	1·29	1·43	1·41	1·14		
Total		5·29	4·98	5·40	5·25	5·25	4·2	4·3

*For households with at least one child under age 5.

(2) Larger families are known to be associated with lower social status. The association between size of family and incidence of enteritis may, therefore, arise from both these factors being correlated simultaneously with poor socio-economic conditions. Equally, the reverse could apply: the real association might be that between enteritis incidence and size of family, and the apparent connexion between social class and enteritis might arise spuriously from the larger families in lower social classes. This triangular effect is difficult to disentangle without a specially-designed factorial experiment.

(3) There is a *prima facie* hypothesis that infection will be more frequent where the number exposed to risk of infection is larger. The larger the household the more likely is infection to be imported into the household, and the more likely is it to be transmitted from case to case. It has been found (Stein, 1952) that tuberculosis mortality is more closely associated with the number of persons per dwelling than with the extent to which those persons are crowded together.

HOUSING CONDITIONS.—The type and density of housing are shown in Table VI and the figures are compared with those of a recent survey in the study area (Gore and Hartston, 1953) and with certain census figures.

TABLE VI
HOUSING CONDITIONS

Type of Accommodation		Total		No Sign of Diarrhoea		Enteritis						Divisions 1 and 2 1952 Survey		London A.C. Census 1951 1% Sample
						Mild		Moderate		Severe				
		No.	Per cent.	No.	Per cent.	No.	Per cent.	No.	Per cent.	No.	Per cent.	No.	Per cent.	
House		40	11	9	24	13	9	12	11	6	8	10	6	
Flat		67	19	8	22	27	19	19	18	13	18	47	26	
Rooms		165	46	16	43	65	45	48	46	36	52	115	64	
Communal		42	12	3	8	15	10	18	17	6	8	—	—	
Residental Nursery		44	12	1	3	25	17	8	8	10	14	—	—	
Other		—	—	—	—	—	—	—	—	—	—	8	4	
Not stated		2	—	—	—	1	—	1	—	—	—	—	—	
Total		360	100	37	100	146	100	106	100	71	100	180	100	
Persons per Room (excluding communal accommodation) ..		2·05		1·44		2·23		1·90		2·14		1·38		0·83
Sanitation	W.C.	358		37		146		106		69				
	Other	2		—		—		—		2				
	Shared	215	60	15	41	88	60	68	64	44	62			35
Washing Facilities	Good	105	31	14	40	36	27	33	33	22	33			
	Adequate	141	42	15	43	56	41	42	43	28	41			
	Bad	91	27	6	17	43	32	24	24	18	26			
	Not stated	23	—	2	—	11	—	7	—	3	—			
Washing-up facilities shared ..		115	34	5	14	51	38	30	30	29	43			†16
Sanitary Defects	Dirty Dustbin ..	1		—		—		—		1				
	Condemned Property	4		—		3		—		—				
	Bad Drains	1		—		1		—		—				

*For households with at least one child under age 5. †Sharing or without a kitchen sink.

With regard to type of accommodation, it will be seen that, compared with average conditions, the families in the study had access to a normal share of the available houses and flats, but that there was an unduly large representation of communally-accommodated families and of infants in residential nurseries. Nearly one-quarter of the cases (86) came from such a source, where, merely on the basis of the numbers at risk, the facility of transmission of infection in the presence of one infecting case is enhanced. Furthermore, excluding the communal accommodation, there was more than average crowding in these homes as measured by the density index of persons per room.

For all the families studied (excluding those in communal accommodation), the average density was 2·05 persons per room, compared with 1·38 for a sample survey of the general population of the area of similar household constitution. The census figure of 0·83 has been inserted for completeness, but it is naturally lower because it relates to the whole population including families without children.

Direct expression of this crowding or of conditions which are likely to be associated with crowding are given by the remaining figures in Table VI. Although actual personal washing facilities were bad in only a quarter of the households, conditions were much worse at "kitchen" level—one-third of the households had to share washing-up facilities with another household, a fraction twice as large as in the county as a whole. With regard to sanitation, nearly two-thirds of the households had to share a water-closet; the proportion in the county as a whole is only half as large. These two factors have a similar action in increasing the likelihood of spread of infection from family to family.

CLEANLINESS IN THE HOME AND CARE OF FOOD.—The health visitors made personal, subjective, assessments of the standard of cleanliness in the homes. A little less than half (41 per cent.) of the households were regarded as barely satisfactory, and one-sixth (17 per cent.) were regardad as bad.

In their attitude towards the care of food, nearly one-half the families were careless or dirty (Table VII). Generally speaking, food was adequately protected against flies, vermin, and dust in some three-quarters of cases (the proportions are remarkably constant in the various groups). Heat was less well guarded against; only one-quarter of families took adequate precautions (of these two-thirds, 18 per cent. of the total, had refrigerators). Evidence of flies was seen in about one-fifth of households, and evidence of mice or other vermin in about the same proportion of cases. There is no control group with which to compare this information. Recalling also that there was no seasonal swing in incidence of admission to hospital, it would not appear that a strong case could be made for suggesting that contamination of food by flies

TABLE VII
CARE OF FOOD*

Factors Examined		Total		No Sign of Diarrhoea		Enteritis					
						Mild		Moderate		Severe	
		No.	Per cent.	No.	Per cent.	No.	Per cent.	No.	Per cent.	No.	Per cent.
Hygiene	Clean	181	54	23	66	76	55	45	47	37	56
	Careless	120	36	11	31	49	36	36	37	24	36
	Dirty	34	10	1	3	13	9	15	16	5	8
	Not stated	25	—	2	—	8	—	10	—	5	—
	Total	360	100	37	100	146	100	106	100	71	100
Protection against	Heat	72	27	5	16	25	25	25	30	17	33
	Dust	208	78	26	81	73	73	66	79	43	83
	Flies	213	79	26	81	76	76	68	81	43	83
	Mice, etc.	190	71	27	84	64	64	64	76	35	67
	Not stated	92	—	5	—	46	—	22	—	19	—
Refrigerator		50	19	3	9	23	23	12	14	10	19
Vermin	Flies	67	20	2	6	29	21	22	23	14	21
	Mice, etc.	75	22	7	20	30	22 .	23	24	15	23

*Percentages are "of stated" cases.

or multiplication of organisms in food due to lack of refrigeration were major aetiological factors.

PET ANIMALS.—There was a cat in 81 households (23 per cent.) and a dog in 33 (9 per cent.). There were three canaries, and no parrots. The normal frequency of pets is not known but these figures would not be regarded as unduly large.

PREPARATION OF FOOD FOR BOTTLE-FED INFANTS.—We have seen that washing-up arrangements, and presumably other kitchen facilities, were shared in one-third of all cases; in fact, for the bottle-fed infants, only 164 (59 per cent.) mothers had separate facilities for preparing feeds. The materials used for bottle-feeds are shown in Table VIII. Three-quarters of the bottle-fed infants were being given dried milks, and the rest, with two exceptions, were having pasteurized or sterilized liquid cow's milk.

TABLE VIII

MATERIALS OF BOTTLE FEEDS

Bottle Feeds		No.	Per cent.
Cows' Milk	Pasteurized	62	22·4
	Sterilized	5	1·8
	Raw	2	0·7
Dried Milk		208	75·1
Total		277	100·0

Details of the methods of preparing feeds are shown in Table IX. In the opinion of the health visitors, bottles were adequately sterilized in little more than half the cases (57 per cent.), though the feeds themselves were boiled in nine-tenths of cases (88 per cent.). There were very few instances (4 per cent.) where milk was prepared for several feeds at one time. On the whole the impression was that the standard of hygiene in bottle feeding could be improved, and that, apart from the presumed inferiority of cow's milk as compared with breast milk, the lack of cleanliness in feeding, particularly in relation to the sterilization of utensils, may have been an aetiological factor in the development of diarrhoea and vomiting.

The health visitor attached to St. Ann's Hospital who investigated a series of admissions to the enteritis unit in more detail reported as follows:

> The care of feeding bottles and teats was poor. Of 112 mothers using bottles, only 34 used any form of detergent to cleanse them; the remaining 78 seemed to think that this would be harmful to the infant, and some used methods of their own for removing milk scum, such as tea leaves, wet newspaper and sand. Three mothers used the Milton technique of disinfection correctly, and two incorrectly; fourteen boiled both bottle and teat after each feed, seventeen boiled them at least once a day, five boiled sometime during the week; 41 relied on domestic scalding only, and 37 made no attempt to disinfect at all. A small proportion of mothers protected the bottle and teat between feeds with a clean cloth or in a special receptacle; the majority stored them in a cupboard or drawer with other crockery; and 37 left them exposed. This poor bottle hygiene was the same in all severity groups, in the group of cases with specific enteritis, in those with parenteral infection, and in the group of infants with no diarrhoea.
>
> Information obtained from mothers of healthy infants from much the same type of homes, showed almost exactly the same picture. Of 159 mothers interviewed, 59 used a detergent and 100 rinsed with plain water only; 59 disinfected adequately once a day, 109 either scalded or made no attempt to disinfect; 114 covered or partially covered bottle and teat, 45 left them exposed between feeds. Of fifteen mothers attempting the Milton technique, fourteen were not carrying it out correctly, using too weak a solution, not covering bottle and teat adequately, or not changing the solution regularly.

TABLE IX

PREPARATION OF FEEDS

Methods		Total		No Sign of Diarrhoea		Enteritis					
						Mild		Moderate		Severe	
		No.	Per cent.	No.	Per cent.	No.	Per cent.	No.	Per cent.	No.	Per cent.
Bottles adequately sterilized		157	57	9	41	64	61	44	52	40	61
Feeds boiled		245	88	22	100	95	90	76	90	52	79
Feeds made up	Individually	265	96	20	91	101	96	82	98	62	94
	For several feeds at once	12	4	2	9	4	4	2	2	4	6
Total Cases		277		22		105		84		66	

MULTIPLE BIRTHS.—There were 29 twins in the series, an incidence of 8·0 per cent., compared with an expectation (on the basis of all maternities in England and Wales in 1950) of 2·4 per cent. This difference is statistically significant, and suggests that twins are more susceptible than infants of single births to attacks of diarrhoea. It may be directly due to the twins' initially smaller size and reduced resources, or indirectly to their being more likely to be bottle-fed.

MEDICAL HISTORY OF INFANTS AND FAMILY MEDICAL BACKGROUND.—Information obtained about other children in the household who had previously suffered from attacks of enteritis is shown in Table X.

A previous attack of enteritis had occurred in children of the same household in a large proportion of cases; in one-third of those with brothers or sisters one of them had been attacked, and in about a half of the households where there were unrelated children such children had been previously affected. If attacks within one month only are considered, these proportions are reduced to one-seventh and one-third respectively, but are still substantial. It seems possible that some of the cases of enteritis in the study can be accounted for either by a common source of infection or by cross-infection from one child to another within the family, but it is not possible to identify such cases with any degree of certainty in a retrospective survey of this kind. The histories of the children in the study are shown in Table XI.

Nearly one-third of the children had a poor health record. One-tenth (10 per cent.) of them had been admitted for other infectious diseases, and a further 11 per cent. had received hospital treatment for non-infectious diseases. One would have expected a sample of children selected on account of hospital admission to be *ex hypothesis*, an unhealthy group, but one would hardly expect such a high incidence of previous hospital admission in children of this age. It suggests that constitutional or environmental defects were coincident with the accident of infection and possibly conditioned the development of disease.

TABLE X
FAMILY HISTORY OF ENTERITIS

Factors Assessed		Total		No Sign of Diarrhoea		Enteritis					
						Mild		Moderate		Severe	
		No.	Per cent.	No.	Per cent.	No.	Per cent.	No.	Per cent.	No.	Per cent.
Brother or Sister	Within 1 month	34	13	—	—	12	11	17	21	5	10
	Earlier	59	22	6	30	20	18	27	33	6	12
Siblings in Household		265	100	23	100	111	100	82	100	52	100
Other Children	Within 1 month	57	34	1	17	28	35	16	36	12	31
	Earlier	29	17	1	17	17	21	8	18	3	8
Other Children in Household		169	100	6	100	80	100	44	100	39	100

TABLE XI
HISTORY OF PATIENTS

Factors Assessed		Total		No Sign of Diarrhoea		Enteritis					
						Mild		Moderate		Severe	
		No.	Per cent.	No.	Per cent.	No.	Per cent.	No.	Per cent.	No.	Per cent.
General Health	Good	251	71	28	78	101	71	74	70	48	69
	Poor	103	29	8	22	41	29	32	30	22	31
	Not assessed	6	—	1	—	4	—	—	—	1	—
	Total	360	100	37	100	146	100	106	100	71	100
Previous Hospital Admission for	Enteritis	36	10	3	8	17	12	14	13	2	3
	Other infection	29	8	4	11	11	8	8	8	6	9
	Other illness	38	11	6	16	16	11	12	11	4	6

TABLE XII

MEDICAL HISTORY OF FAMILIES

Factors Assessed		Total		No Sign of Diarrhoea		Enteritis					
						Mild		Moderate		Severe	
		No.	Per cent.	No.	Per cent.	No.	Per cent.	No.	Per cent.	No.	Per cent.
General Health	Good	277	82	28	82	107	79	87	85	55	83
	Poor	61	18	6	18	29	21	15	15	11	17
	Not assessed	22	—	3	—	10	—	4	—	5	—
	Total	360	100	37	100	146	100	106	100	71	100
Severe Illnesses	Tuberculosis	32	9	5	14	10	7	14	13	3	4
	Other infectious disease ..	38	11	4	11	19	13	8	8	7	10
	Non-infectious disease ..	54	15	7	19	21	14	18	17	8	11

Table XII shows that the medical histories of the families, as distinct from the children of the study, were also poor, one-sixth having a poor health record. The incidence of histories of tuberculosis (9 per cent.) was not significantly higher than the normal figure (8 per cent.) for the area (Gore and Hartston, 1953), but considerable numbers of severe infectious and non-infectious diseases were encountered. We have already discussed the lower than average social conditions of these families; even the very limited information of the medical histories of these children and their families serves to illustrate the simple tenet of social medicine that families with socio-economic disadvantages may be an easier prey to infection and disease. Further, in a quarter of all cases admitted to the enteritis unit at St. Ann's from their own homes, there had been some recent domestic upset, which meant either that the infant had been looked after by someone other than his mother or that he had been exposed to a greater risk of infection. In most instances the upset was due to illness of the mother, illegitimacy, separation of parents, imprisonment of father, or holidays. In some instances illness or feeding problem in the infant had led to his admission to hospital or nursery. These upsets were marked in the group not showing signs of parenteral infection and these cases constituted a large proportion of the group having a definite history of contact with cases of diarrhoea or with an institution or other infant community. Illegitimacy appeared to be a problem in so far as breast-feeding in these cases had usually been abandoned from financial necessity, and hazards of exposure to infection thereby increased. This underlines the difficulty of trying to isolate specific aetiological factors in enteritis from the complex environment of such families.

MODE OF ONSET OF DIARRHOEA AND OTHER SYMPTOMS AND INCIDENCE OF PARENTERAL INFECTION.—Table XIII (overleaf) shows the mode of onset of the diarrhoea for which the children were admitted to hospital.

In two-thirds of the cases the onset of diarrhoea was abrupt, even in those in which there was no diarrhoea after admission to hospital, in one-quarter the onset was insidious, and in one-seventh the diarrhoea had been recurrent. In most, but not all, the diarrhoea itself was the first symptom. In about one-third there were signs of parenteral infection at the time of onset of diarrhoea and vomiting. Table XIII shows that symptoms of parenteral infection were present in more of the cases showing no sign of diarrhoea in hospital than in those with severe diarrhoea.

Contact with parenteral infection within 2 weeks of onset of diarrhoea was established as follows:

Coryza	32 per cent.	Influenza	3 per cent.
Sore throat	5 per cent.	Tuberculosis	1 case only.

A history of dietetic disorder preceding the onset of diarrhoea and vomiting was found in only a few cases. The dietetic factor was rarely found alone, some cases having parenteral infections and others having been recently in contact with diarrhoea or with an infant community.

CONTACT WITH DIARRHOEA (Table XIV, overleaf).—In almost half the cases recent contact with a case of diarrhoea could be established, most of them in children under 2 years of age. The degree of contact was consistent in the groups and was no greater in the severe than in the mild cases.

Among cases admitted to the enteritis unit at St. Ann's, a history of close contact with illness in the 2 weeks before onset was found most frequently

TABLE XIII
CIRCUMSTANCES OF ONSET

Factors Assessed		Total		No Sign of Diarrhoea in Hospital		Enteritis					
						Mild		Moderate		Severe	
		No.	Per cent.	No.	Per cent.	No.	Per cent.	No.	Per cent.	No.	Per cent.
Mode of Onset of Diarrhoea and Vomiting	Abrupt	220	62	23	65	86	61	64	61	47	67
	Insidious	88	25	9	26	34	24	28	27	17	24
	Recurrent	44	13	3	9	22	15	13	12	6	9
	Not known	8	—	2	—	4	—	1	—	1	—
	Total	360	100	37	100	146	100	106	100	71	100
Onset Associated with	Diarrhoea	312	87	31	84	126	86	93	88	62	87
	Vomiting	241	67	27	73	87	60	73	69	54	76
	Coryza	83	23	17	46	32	22	24	23	10	14
	Otorrhoea	19	5	1	3	11	8	5	5	2	3
	Bronchitis	18	5	1	3	8	5	6	6	3	4
	Pneumonia	6	2	—	—	2	1	3	3	1	1
	Other symptoms	165	46	24	65	57	39	51	48	33	46

TABLE XIV
CONTACT WITH OTHER CASES BEFORE ONSET

Factors Assessed		Total		No Sign of Diarrhoea		Enteritis					
						Mild		Moderate		Severe	
		No.	Per cent.	No.	Per cent.	No.	Per cent.	No.	Per cent.	No.	Per cent.
Children Under 2	Within 2 weeks of onset	88	24	2	5	37	25	29	27	20	28
	Over 2 weeks previously ..	15	4	—	—	10	7	4	4	1	1
Children Over 2	Within 2 weeks of onset ..	55	15	5	14	18	12	24	23	8	11
	Over 2 weeks previously ..	7	2	—	—	4	3	3	3	—	—
Adults	Within 2 weeks of onset ..	19	5	4	11	8	6	6	6	1	1
	Over 2 weeks previously ..	2	1	—	—	1	1	1	1	—	—
Total Cases		360		37		146		106		71	

in cases coming from another institution. Even in those coming from their own homes, with no history of contact with diarrhoea or parenteral infection, a substantial proportion had been resident in a hospital or other institution within 2 weeks of onset.

GENERAL CONTACT WITH OTHER PERSONS (Table XV, opposite).—This information is of epidemiological value in itself, since it shows how many people a child of under 2 years of age can meet in the space of a fortnight. Only one-sixth had no contact outside their own family. Nearly one-half (41 per cent.) were in contact with adults or children of other families. Only a very small number had been to day nurseries. A quarter of the cases (23 per cent.) came from communal accommodation or from residential nurseries where the opportunities for the spread of infection are inevitably enhanced. One in four of the children (24 per cent.) had been attending a welfare centre, and a further fifth (21 per cent.) had been attending a hospital outpatient department. Clearly, apart from traced contact with infection, there was here a large reservoir from which infection could have come.

BACTERIOLOGY.—Laboratory investigations were not uniformly pursued in all hospitals, and this section of the study is, therefore, based on data from St. Ann's Hospital only. At this hospital, rectal swabs were taken from all 220 cases, and, with only eight exceptions, they were taken on admission.

The enteral pathogens found are listed in Table XVI (opposite).

Rectal swabs from 57 infants (three of whom were admitted twice) were also examined for the serological types *Bact. coli* 0 111 and *Bact. coli* 0 55; the first was isolated from six and the second from two infants.

TABLE XV
CONTACT DURING 2 WEEKS BEFORE ONSET

Type of Contact		Total		No Sign of Diarrhoea		Enteritis					
						Mild		Moderate		Severe	
		No.	Per cent.	No.	Per cent.	No.	Per cent.	No.	Per cent.	No.	Per cent.
Home	Own Family Only	56	15	6	16	18	12	15	14	17	24
	Other Families	148	41	14	38	67	46	44	42	23	32
	Child-minder	9	3	1	3	4	3	2	2	2	3
	Foster Parents	4	1	—	—	2	1	—	—	2	3
Institution	Day Nursery	21	6	1	3	12	8	5	5	3	4
	Rest Centre	42	12	3	8	15	10	18	17	6	8
	Residential Nursery or Hostel	41	11	1	3	22	15	8	8	10	14
	Child Welfare Centre ..	88	24	12	32	28	19	27	25	21	30
	Hospital (Out-patient Dept.)	35	10	2	5	15	10	10	9	8	11
	Hospital (Children's Dept.)	38	11	5	14	8	5	14	13	11	15
Other		—	—	3	8	4	3	3	3	3	4
Total Cases		360		37		146		106		71	

TABLE XVI
ENTERAL ORGANISMS (OTHER THAN *B. COLI*)

Organism		Total		No Signs of Diarrhoea		Enteritis					
						Mild		Moderate		Severe	
		No.	Per cent.	No.	Per cent.	No.	Per cent.	No.	Per cent.	No.	Per cent.
Salmonella	Typhi-murium	5	4	—	—	2	6	2	4	1	2
	Enteritidis	1		—		1		—		—	
	Morb. bovis	3		—		2		1		—	
Shigella		17	8	1	3	11	14	4	6	1	2
Lamblia		2	1	—	—	2	3	—	—	—	—
Total Cases		220	100	30	100	79	100	70	100	41	100

ORIGIN OF HOSPITAL ADMISSIONS.—The cases surveyed in this study were selected by virtue of hospital admission. It is of interest, therefore, to examine the origin of the certificate of diarrhoea and vomiting by which hospital admission was gained (Table XVII, overleaf).

Nearly two-thirds (60 per cent.) came from general practitioners, just over one-fifth (21 per cent.) came from out-patient departments, and a little more than one twentieth (6 per cent.) were transferred from in-patient departments; 43 cases (12 per cent.) came from residential nurseries. The distribution of origin of case was not related to severity of illness.

CONCLUSIONS

This survey was restricted to infants admitted to hospital for diarrhoea and vomiting and thus to a selected group of the total cases of enteritis occurring in the area of study. They were selected on the grounds of severity and socio-economic factors also influenced admission to hospital. Nevertheless, the pattern, as it has emerged, is one of infection spreading among families of lower than average social conditions with poor medical records. The facility of transmission of infection was enhanced by a number of inter-related conditions. The large families, and in many cases the communal accommodation, associated with their lower social level, presented larger numbers at risk and a greater degree of herding; many were forced to share water closets and washing-up facilities. Many of the babies were underweight at birth, and, whether associated with this condition or not, most were bottle-fed. The standard of cleanliness in bottle-feeding was low though no worse than in a sample

TABLE XVII
ORIGIN OF CERTIFICATE OF DIARRHOEA AND VOMITING

Source		Total		No Sign of Diarrhoea		Enteritis					
						Mild		Moderate		Severe	
		No.	Per cent.	No.	Per cent.	No.	Per cent.	No.	Per cent.	No.	Per cent.
Out-patient Dept.	General Hospital	48	13	6	16	21	14	14	13	7	10
	Children's Hospital ..	29	8	2	5	7	5	15	14	5	7
In-patient Dept.	General Hospital	17	5	3	8	3	2	7	7	4	6
	Children's Hospital ..	3	1	—	—	2	1	—	—	1	1
Residential Nursery		43	12	1	3	24	16	8	8	10	14
General Practitioner		215	60	25	68	87	60	62	58	41	58
Welfare Centre		5	1	—	—	2	1	—	—	3	4
Total Cases		360	100	37	100	146	100	106	100	71	100

of healthy infants. There were few specific feeding disorders. Some two-thirds of the cases were considered to be due to alimentary infections, and in these there was a high degree of known contact with other cases of diarrhoea; one-third of the cases could be regarded as influenced by parenteral factors, and had signs or symptoms of parenteral infection at the time of onset of the diarrhoea.

The facts presented suggest that a reduction in the incidence of enteritis might be expected from:

(*a*) Improvement in housing, directed particularly towards avoiding the sharing of sanitary and kitchen accommodation.

(*b*) A study of the conditions influencing the rate of breast-feeding, with a view of increasing maintenance of breast-feeding.

(*c*) Better education on the hygiene of preparation of bottle feeds.

(*d*) Treatment as a priority class by health visitors of families with poor health records.

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REFERENCES

Douglas, J. W. B. (1950). *J. Obstet. Gynaec. Brit. Emp.*, **57**, 335

Gore, A. T., and Hartston, W. (1953). *Monthly Bull. Minist. Hlth, Lond.*, **12**, 131.

Stein, L. (1952). *British Journal of Social Medicine*, **6**, 1.

Brit. J. prev. soc. Med. (1953), 7, 205–210

MENTAL HEALTH IN RELATION TO THE LABOUR TURNOVER OF UNSKILLED WORKERS IN A LARGE INDUSTRIAL ESTABLISHMENT

BY

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Study of the relationship between mental health, psychological handicap, and industrial conditions has developed considerably in importance during the last 5 years. Wartime investigations connected with the occupational adaptation of discharged service men were described by Lewis and Slater (1942), Lewis (1943), and Guttman and Thomas (1944), and the findings of Fraser (1947) on the incidence of neurosis in factory populations and its association with absenteeism are well known. The effects of such disability on working efficiency are by no means clear. Studies in factories (Markowe and Barber, 1952; 1953) have not confirmed the widely held notion that individuals who have a neurotic handicap often produce less than the average worker, though they showed that those with poor intelligence tended to have a lower output.

It has frequently been asserted that neurotic workers are more prone to change their job than those enjoying normal mental health. Reasons for leaving employment may, however, be social and economic as well as psychological (Long, 1951). In the present inquiry an attempt is made to relate labour turnover to mental health, by examining the association between length of stay in a new job and the incidence of neurotic disability. The study is based on unskilled workers in two large factories; men and women are considered separately.

Material

Men.—This part of the study was carried out in an extensive medium and heavy engineering factory on the fringe of Manchester. Preliminary meetings were held with management, foremen, and shop stewards in order to explain the purpose of the study and obtain their co-operation and assistance; it would otherwise have been impossible to approach individual workers directly and ask them to take part in the investigation. An industrial dispute between the Engineering Union and employers in the North West Region delayed the project by some months, since the normal pattern of entry and exit in the industry was thereby affected. The attitude of industrial workers to the study would also have been suspicious or hostile at that time.

A random sample was taken, consisting of 100 unskilled male workers accepted for employment at the factory between March and November, 1951. The sample was selected by using tables of random numbers on the weekly lists of "starters" supplied by the labour office. The selection was in no way related to the department or actual job to which workers were assigned. Two men refused to be interviewed; they were replaced in the sample.

The workers were assigned to unskilled tasks in different departments; these included automatic machine tending, non-automatic machine tending, inspection, manual labour, and work on electrochemical processes or special constructions. Excluded were workers below the age of 23 (and therefore still liable to national service), and foreigners who might have found language difficulties in the tests.

Women.—This part of the enquiry was carried out in a large light engineering factory, also on the fringe of Manchester, employing mainly female labour. Fresh workers were constantly needed and therefore an adequate sample was readily obtained. Because of the tradition of workers' welfare in the firm, the women were ready to co-operate, and the research had the initial approval of the management and shop stewards.

A random sample was taken, consisting of 100 unskilled female workers accepted for employment at the factory between March and November 1951. The workers were engaged in the machining or assembly of small components and the sorting, grading, and packing of small parts. Such work generally demands finger dexterity and patience, but rarely strenuous exertion; facility in it could usually be achieved after a short training.

Methods

Each worker was interviewed clinically during the first week of employment. Height, weight, visual acuity, and haemoglobin were measured. A medical questionnaire followed, and then a test of general mental ability. Special tests of blood pressure, ischaemic endurance (described below), and exercise tolerance (Behnke) were

administered. Each individual's life history, health, and occupational record were ascertained, and his psychological handicaps of personality assessed at the time of the interview and during the previous 12 months. He was then rated on five-point scales for mental health and personality traits.

Recent mental health ranged from Rating 1 (healthy, mature, vigorous, and well-integrated) to Rating 5 (definite disabling sickness, mainly of moderate severity, requiring treatment). Ratings 2 and 3 covered the rest of the "normal" range; Rating 4 included minor states of ill-health which, although they did not materially incapacitate the individual or oblige him to seek treatment, interfered with his domestic, social or working life. Those with Ratings 1, 2 and 3 were considered to be "normal". Those with Ratings 4 and 5 were considered to be "psychologically handicapped"; they were neurotic or emotionally unstable.

The personality traits assessed were Resolution, Affective Lability (emotional instability), Extraversion, Emotional Maturity, Obsessionality, and Conscientiousness; they were operationally defined.

In addition to clinical assessment of intelligence or dullness, a pure test of general mental ability (the Dominoes Test) was administered under standard conditions. This is a non-verbal test, consisting of 48 items, which has been well standardized and validated on large groups of national service men taken into the Army.

Finally, blood pressure estimation was followed by inflation of the cuff to a pressure of 250 mm. Hg., while the subject was instructed to clench and open his fist at the rate of once per second for as long as he could bear it, or up to 5 minutes. In this ischaemic state responses ranged from no discomfort to acute distress; the majority experienced much discomfort. The length of time in seconds was the score of "endurance".

The occupational record obtained during the interview included data about the duration of each past job and reasons for leaving it.

Each subject was "followed up" for a period of 6 months. The nature of his or her work was ascertained, as well as his adjustment to it, his absences, and whether he left during this period. "Leavers" were defined as those who left within 6 months of being taken on. During the period of the study no one was dismissed. Comments were obtained from the subjects' immediate supervisors about their attitude to the job.

Certain general considerations restricted the clinical interview. First, absence from work lowered the subject's earnings, as this period was not covered by payment on the firm's basic rates. Secondly, if (as was usual) the worker felt "healthy", he failed to see the need for the physical examination, tests, and survey of his history, so that the tests and physical appraisal had to be brief.

Results

Samples

Men.—Ages ranged from 20 to 62, with a mean of 39·35 (standard deviation 11·3); there were nearly as many over 45 as under (25 were between 20 and 29; 33 were between 30 and 44; and 42 were 45 or over). Of the 100 men, 78 were married, and only sixteen of these had no children.

Normal mental health was found in 57 of whom 41 were entirely free from symptoms; 43 were psychologically handicapped, nine being moderately and 34 mildly affected (see Table I). Their handicaps included anxiety states, depressive states, emotional instability, dullness, and feeble-mindedness. Nevertheless only a few of these 43 men were under medical treatment.

Intelligence scores as measured by the Dominoes Test ranged from 5 to 35 points (maximum possible was 48), with a mean of 22·71 and standard deviation of 6·93. This corresponds to a mean IQ of 91; this equivalence is not derived from direct standardization and must therefore be considered approximate.

Women.—The women formed a cross-section of the working community during a year of full employment. They were heterogeneous in most respects, yet uniform in the desire to earn, often in order to augment their family budget.

Ages ranged from 15 to 58, with a mean of 32·82 (standard deviation 10·29), with a majority below 45 (38 were between 15 and 29, 47 between 30 and 44, and 15 were 45 or over). Of the 100 women, 73 were married, and only sixteen of these had no children.

Normal mental health was found in 45, of whom only 21 were entirely free from symptoms; 55 were psychologically handicapped, of whom nineteen were moderately and 36 mildly affected (see Table I). The handicaps included anxiety states with and without somatic symptoms, hypochondriasis, oversensitive and suspicious traits, emotional instability, and inadequate personalities. The married women showed a higher incidence of psychological ill-health than the single.

Intelligence scores on the Dominoes Test ranged from 7 to 36 points, with a mean of 21 (standard deviation 6·43), corresponding to an IQ of 89 (see above under *Men*).

Socio-Medical Factors in Relation to Leaving

Within 6 months of having been taken on, 21 men and 52 women had left work.

Health ratings, personality traits, and biographical data were tabulated against leaving, and in some cases against psychological handicap. Standard tests of significance showed few statistically significant associations. Thus a clear association was found in the men between leaving and psychological handicap, past employment record, affective lability,

and parental deprivation, and in the women between leaving and past employment record, certain personality traits, and performance on the ischaemia test. The remaining comparisons, though not significant at the conventional level did not indicate complete lack of association. Owing to the impossibility of controlling independent variables in the social occupational field, the attenuation of association is to be expected. In these circumstances probability levels between 0·05 and 0·2 may not be arbitrarily dismissed.

Psychological Handicap

Men.—Fourteen of 21 men who left were psychologically handicapped, as compared with 29 of 79 who stayed. It would, however, be unsafe to attribute a great deal to this difference, since only two of the fourteen who left were moderately handicapped, while of the 79 seven were moderately neurotic. Nevertheless the difference is significant ($\chi^2 = 6{\cdot}07$, $P < 0{\cdot}02 > 0{\cdot}01$). Twelve of the 34 mildly handicapped men left, and 22 stayed.

Women.—32 of 52 women who left were psychologically handicapped, as compared with 23 of 48 who stayed ($\chi^2 = 1{\cdot}87$, $P < 2 > 0{\cdot}1$). Although the difference is not significant, it is in the same direction as in the male group. Fourteen of the nineteen moderately handicapped women left, as against five who stayed. Eighteen of the 36 mildly handicapped left and eighteen stayed.

Personality Traits

Men.—Those with "affective lability" showed a significant association with leaving ($\chi^2 = 5{\cdot}69$, $P < 0{\cdot}05$, $> 0{\cdot}02$). Men with stable temperament and steady emotional state, whether phlegmatic or pessimistic, tended to stay on the job.

Women.—Conscientiousness and obsessionality were both found to be significantly related to staying on the job ($\chi^2 = 5{\cdot}69$, $P < 0{\cdot}02$, $> 0{\cdot}01$ for conscientiousness; $\chi^2 = 3{\cdot}93$, $P < 0{\cdot}05$, $> 0{\cdot}02$, for obsessionality). Among the women, the extraverts showed a greater tendency to leave.

There was no association between leaving and either maturity or resolution in the men or women.

Ischaemia Test of Endurance.—The time of endurance in seconds of obliterative ischaemia of the right upper arm was noted and the means calculated for those who left and for those who stayed.

Men.—Those who left had a mean score of 184·7 seconds, as compared with 198·59 seconds for those who stayed. Standard error of the difference 19·216.

Women.—Those who left had a mean score of 146 (standard deviation 4·79), and those who stayed a mean score of 175 (standard deviation 80·64). Standard error of the difference 11·66. It should be noted that the variation about the mean, while small in the case of those who left (indicating a relatively compact group) is considerable for those who did not leave (possibly due to the many reaching an arbitrary maximum score of 300 sec.).

Age.—There was no significant relationship between age and leaving, although in the men there were more psychologically handicapped in the younger age group. In the women there was no significant relationship between age and psychological handicap; more of the younger women tended to leave, but the trend was not significant.

Mental Ability.—There was no association between leaving and mental ability, either in the men or in the women.

Marital Status

Men.—The married workers compared with single were fourteen to seven in the leavers, and sixty-four to fifteen in the non-leavers; thus the married appeared to be less disposed to leave, but the difference is not statistically significant. The married with children exceeded the childless to a greater degree among leavers than among non-leavers, but the difference is not significant.

Women.—Of those who left, 42 were married as compared with ten single; of those who stayed, 31 were married and seventeen single. The difference is not significant ($X^2 = 3{\cdot}33$, $P < 0{\cdot}05$, $> 0{\cdot}1$).

Past Changes of Employment.—The changes in employment under consideration in this paper cover the years 1945–51, a period of full employment coupled with the partial removal of the Essential Works Order. This period was chosen in order to secure:

(*i*) a defined period sufficiently short for reliable statements and recollections,

(*ii*) a period within which earnings and opportunities of employment were stable and comparable.

The period of sampling for this study escaped the subsequent recession and redeployment in the textile industry—an unexpected development which would have affected the turnover of labour, and the proportions of normal and handicapped individuals in the samples.

Men.—A comparison between those with a history of one to three changes of job in this period and those with a history of four or more jobs,

showed that the psychologically handicapped had been through more jobs than had the normal group ($\chi^2 = 4{\cdot}949$, $P < 0{\cdot}05$, $> 0{\cdot}02$). As far as the present group of leavers was concerned, seven of the 21 had a history of four or more changes of job in the period 1945 to 1951.

Women.—Those with six and more job changes included thirteen handicapped and four normal; those who had had not more than five jobs included 42 handicapped and 41 normal. ($\chi^2 = 3{\cdot}81$, $P = 0{\cdot}05$). As far as the present group of leavers was concerned, ten of the 52 had a history of six or more changes of job in the period 1945–51, and thirty of the 52 had had four or more changes.

These findings support the assumption that persons with psychological handicaps tend to change their jobs more often than do those in the normal range.

Parental Deprivation

Men.—Absence of one or both parents during childhood, before the worker had left school, whether due to death, desertion, or divorce, was found to be associated with leaving ($\chi^2 = 4{\cdot}627$, $P < 0{\cdot}05$, $> 0{\cdot}02$). Out of the total sample of 100, 37 were found to have been so deprived; this is a considerable proportion in any socio-economic group.

Women.—The corresponding number was thirty, and was not significantly associated with leaving.

Industrial Factors in Relation to Leaving

Men.—There was no significant relationship between leaving and the size of the labour force in each department, or the nature of the work. Within the labouring group of sixty, however, the handicapped were in a majority among the leavers (nine out of twelve), ($\chi^2 = 6{\cdot}817$, $P < 0{\cdot}01$, $> 0{\cdot}001$).

Women.—There was no significant relationship between leaving and the size of the labour force in each department, the nature of the work, predominance of women over men, or wholly female staffing. Nor was there a significant relation between any of these factors and psychological handicap.

Length of Stay

Men.—Those who left in the first 10 weeks were normal or handicapped in equal numbers, whereas those who left between 11 and 26 weeks after starting were all handicapped. On the average, the handicapped left after 10·4 weeks, as compared with 4·7 weeks for the normals who left ('t' is significant at 0·05 level).

Women.—The handicapped appeared to leave either early in their occupational encounter or after many weeks; the normal appeared to leave early or to stay for more than 6 months. On the average, the handicapped left after 11 weeks, as compared with 10 weeks for the normals who left. As the distribution was not normal, 't' test was not applicable. ($\chi^2 = 0{\cdot}98$, $P > 0{\cdot}3$).

Discussion

The high incidence of neurosis in the samples is striking, particularly in the female group (Table I, opposite).

The figures are higher than those observed by Fraser (1947) in a comparable population, though if the non-leavers alone are considered, the disparity is less (Table II, opposite). Many of the early leavers may have eliminated themselves from his population, although they were subject to the Essential Works Order. It is hardly likely that the disparity arises through different criteria of neurosis, since in earlier studies in the light engineering industries we have found figures for the prevalence of neurosis very close to his.

The lesser frequency of leaving among men 21 per cent.) than among women (52 per cent.) indicates a sex difference in occupational behaviour. This phenomenon is well known: thus, for the period March to November, 1951, the electrical machinery industry had a national labour turnover of approximately 16 per cent. for males and 21 per cent. for females (Ministry of Labour Gazette, 1952).

It is not easy to see why men with more severe neurotic disorder tended to stay in their jobs, while those who left were either healthy or had only minor neurotic complaints. Neurotic persons are generally expected to show signs of maladjustment, if at all, soon after entry into a new field of work; experience in the armed services during the war was in conformity with this. But in civil life the anxious neurotic imperils his precarious feeling of security, it may be supposed, if he leaves a newly acquired employment. For women, however, this can be only one factor, as fourteen of the nineteen severely handicapped left within 6 months, and the more severe their neurosis, the sooner they tended to leave.

There was a tendency for marriage to be associated, in the women, with leaving. This may be accounted for by the additional stresses experience by married women when employed in factories. The domestic needs of husband and home, problems of the family

TABLE I

GENERAL DISTRIBUTION OF GROUPS BY MENTAL HEALTH RATINGS AND CONTINUANCE IN EMPLOYMENT

Sex		Normal				Psychologically Handicapped			Grand Total
		1	2	3	Total	4	5	Total	
Male	Leavers	1	5	1	7	12	2	14	21
	Non-leavers	13	22	15	50	22	7	29	79
	Total	14	27	16	57	34	9	43	100
Female	Leavers	3	8	9	20	18	14	32	52
	Non-leavers	3	7	15	25	18	5	23	48
	Total	6	15	24	45	36	19	55	100

Male: For the totals χ^2 6·075 $P < 0{\cdot}02 > 0{\cdot}01$
Female For the totals χ^2 1·87 $P < 0{\cdot}2 > 0{\cdot}1$

TABLE II

PERCENTAGE INCIDENCE OF NEUROSIS COMPARED WITH RESULTS OF PREVIOUS INVESTIGATIONS

Author	Date	Sex	Degree of Neurosis	
			Mild	Definite
Fraser	1947	Male	19·7	8·5
		Female	37·5	10·4
Markowe and Barber	1953	Male	34	9
		Female	36	19

budget, and concern over the care of children, by neighbours or in nurseries, all add to the psychological and physical demands of work in the factory. Many of the married women declared that they were not fatigued by the job but were exhausted at the end of the day after also completing their domestic chores.

It was not surprising that general mental ability showed no relationship to leaving. The types of unskilled work in question do not call for much intelligence. Many dullards and high-grade defectives are accepted by employers as adequate for such work, and various studies have demonstrated that they can adjust satisfactorily (Tizard and O'Connor, 1952).

One of the most significant differences between women leavers and non-leavers was found in the ischaemia test of endurance. While the ability to continue muscular activity in spite of painful discomfort must be affected by physiological as well as psychological factors, in this experiment the varying performance of the women may be assumed to reflect, under conditions of equal motivation, qualities of persistence and determination in the face of difficulty. Such qualities could be highly relevant to the question whether a worker will continue at his employment, whatever the adverse forces. The findings in the men, however, speak against this suggestion; they showed no clear difference between leavers and non-leavers in the endurance of pain in the ischaemic test. The matter calls for fuller study.

Occupational records of both men and women showed that those with neurotic handicaps had changed their employment in the past more frequently than the remainder, although the Association is not however confirmed in this study of 6 month's employment of women workers.

The jobs to which workers are allotted broadly depand upon the demand for labour from departments and in the case of the women upon the empirical recommendations of the training school through which most of them pass. When there is a greater demand for some product, *e.g.* radio sets for the open market or products for the defence programme, the department concerned becomes more willing to carry workers with a psychological handicap such as abnormal anxiety, irritability, or depression.

These findings were obtained in two large factories of a single company in an extensive industrial area. Factories with not less than 250 workers (those here studied each employ 2,000) comprise, in fact, only some 10 per cent. of manufacturing firms, and cover a third of such employment. Therefore, while this study is conclusive in some directions for large employers, it would be unsafe to assume that the present conclusions would be applicable to the wide range of smaller establishments.

SUMMARY

(1) A random group of 100 men was selected from newly engaged unskilled workers accepted for employment at an engineering factory between March and November, 1951. A similar group of 100 women was selected from workers accepted for

employment at a branch factory in the same district during the same period.

(2) Each subject was interviewed clinically in the first week of employment and rated on 5-point scales for mental health and personality traits; tests of mental ability and ischaemic endurance were given, and data obtained about past employment. Each subject was "followed up" for 6 months, and his continuance in the job related to these data. Twenty-one of the men, and 52 of the women left within 6 months of starting the job.

(3) Forty-three of the men and 55 of the women had neurotic symptoms. Of the 21 men who left, fourteen had neurotic symptoms. Of the 52 women who left, 32 had neurotic symptoms.

(4) Seven of the 21 men and thirty of the 52 women had a history of four or more changes of job between 1945 and 1951.

(5) Neurotic men who left did so, on the average, after 10 weeks, compared with 4 weeks for the mentally healthy. There was no significant difference between the non-neurotic and neurotic women in this respect. The more severe the neurotic disability of men who left, the longer they stayed before leaving. Among women the more neurotic left earlier.

6) No association was found between leaving and

(*a*) general intelligence (both sexes);
(*b*) age (men).

(7) Scores of the ischaemia test of endurance were different for women who left and for women who stayed.

(8) Although no association was found between leaving and size of department or type of work, there was a highly significant relation between handicap and leaving in the "labouring" group of sixty men.

Acknowledgments for both studies are made to Professor Aubrey Lewis, Honorary Director of the Unit, for guidance and criticism throughout, to Dr. V. Norris for statistical advice, to our colleague Dr. P. Venables for invaluable assistance in the compilation of the data, and to the Management and Personnel Department of the factories concerned for their lively co-operation.

REFERENCES

Fraser, R. (1947). "The Incidence of Neurosis Among Factory Workers". Rep. industr. Hlth Res. Board., (No. 90) Lond.
Guttman, E., and Thomas, E. L. (1946). "A Report on the Re-adjustment in Civil Life of Soldiers Discharged from the Army on Account of Neurosis". Report No. 93, Min. of Health. H.M.S.O., London.
Lewis, A. J. (1943). *Lancet*, **1**, 167.
—— and Slater, E. (1942). *Ibid*, **1**, 496.
Long, J. R. (1951). "Labour Turnover Under Full Employment". Studies in Economics and Society, Monograph A2. University of Birmingham.
Markowe, M., and Barber, L. E. D. (1952). *Brit. J. industr. Med.*, **9**, 221.
—— and —— (1953). *Ibid.*, **10**, 125.
Ministry of Labour Gazette (1952). **60**, 286.
Tizard, J., and O'Connor, N. (1952). *Lancet*, **2**, 620.

Brit. J. prov. soc. (1953), **7**, 211–219

ANENCEPHALUS, SPINA BIFIDA, AND HYDROCEPHALUS

INCIDENCE RELATED TO SEX, RACE, AND SEASON OF BIRTH, AND INCIDENCE IN SIBLINGS*

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For the congenital malformations, as for many other non-communicable diseases and defects, the collection of epidemiological knowledge based on statistical procedures is just beginning. A comprehensive investigation of the incidence of malformations of the central nervous system in Birmingham, England, and the relationship of these malformations to certain epidemiological variables, has recently been reported by Record and McKeown (1949; 1950a,b; 1951) and McKeown and Record (1951). With the object of determining the applicability of these authors' results to areas other than England and Wales, a somewhat similar, retrospective investigation was undertaken in Rhode Island, U.S.A. This communication describes the findings of this investigation, with the exception of data relevant to the association with maternal age and parity, which is reported separately (Ingalls, Pugh, and MacMahon, 1953).

MATERIAL

The study is concerned with all cases of anencephalus, spina bifida, encephalocele, and hydrocephalus born in the five largest maternity units in the state of Rhode Island during the years 1936–52. For the years 1936–49 births in these hospitals constituted 67 per cent. of all births in the state. For the years 1950–52, the 1949 level of 74·4 per cent. was almost certainly maintained, but vital statistics are not yet available.

Information about the total population of births was obtained from monthly hospital reports and from a sample group assembled by taking every hundredth name from the delivery-room registers of the same hospitals. The registers list chronologically all births occurring in the hospital. For a fuller description of the comparison group, the reader is referred to the discussion of maternal age and birth rank (Ingalls and others, 1953).

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INCIDENCE

The incidence for the three main diagnostic groups in 4-yearly periods is given in Table I. Anencephalus with spina bifida is included with "anencephalus", and spina bifida with hydrocephalus and also encephalocele is grouped under "spina bifida". The first grouping is adopted since, while no note may be made of the presence of spina bifida in an anencephalic baby, it seems less likely that a baby with both anencephalus and spina bifida will be diagnosed as spina bifida. It may well be that the incidence of spina bifida in patients with anencephalus is higher than that here recorded. Spina bifida with hydrocephalus is classified with spina bifida, since many subjects who will develop head enlargement do not exhibit it at birth. The diagnostic data recorded failed to distinguish clearly between encephalocele and cervical meningocele. Very few of these subjects underwent *post-mortem* examination.

For the group as a whole, incidence was highest in the period 1940–43 and lowest in the years 1948–52. Although the highest incidence for anencephalus and hydrocephalus occurred in the years 1944–47, as against 1940–43 for spina bifida, incidence is lowest in 1948–52 for each of the three malformation groups.

TABLE I

INCIDENCE (PER 1,000 TOTAL BIRTHS) OF CENTRAL NERVOUS MALFORMATIONS IN FIVE RHODE ISLAND MATERNITY UNITS

Years	Total Number of Births	Incidence and Numbers of Malformations							
		Anencephalus		Spina Bifida		Hydrocephalus		Total	
		Number	Per mille	Number	Per mille	Number	Per mille	Number	Per mille
1936–39	32,375	47	2·01	59	2·52	22	0·94	128	5·47
1940–43	33,139	71	2·14	120	3·62	30	0·91	221	6·67
1944–47	47,030	107	2·28	116	2·47	52	1·11	275	5·85
1948–52	65,110	101	1·55	131	2·01	48	0·74	280	4·30
1936–52	168,654	326	1·93	426	2·53	152	0·90	904	5·36

The Figure shows the data by single years, and demonstrates a more or less consistent decline from 1942, the peak year, to 1952. Remarkably similar data were presented for these same malforamtions in England by MacMahon, Record, and McKeown (1951). Minor differences between the trends noted in England and in Rhode Island include: an earlier peak of incidence in England (1940–41 as against 1942–43), a somewhat sharper fall in incidence in the British material, and absence of decline in incidence of hydrocephalus in Birmingham.

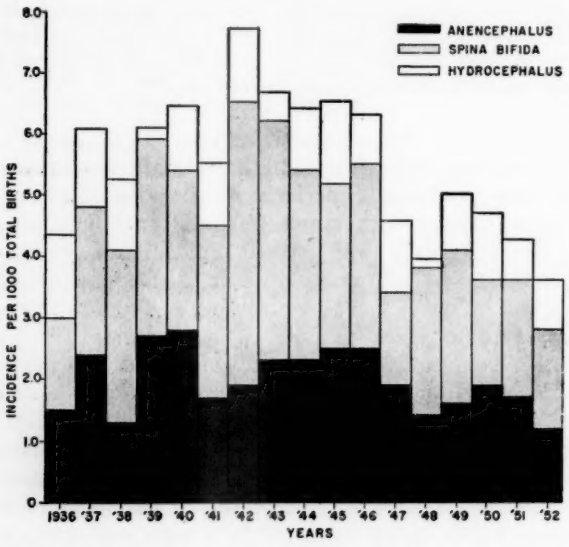

Figure.—Incidence of malformations of the central nervous system in Providence, Rhode Island, 1936–52.

SEX RATIO.—Sex ratios of individual malformations are given in Table II, which exhibits the well-established predominance of females in anencephalus and spina bifida and of males in hydrocephalus.

TABLE II
SEX RATIO

Malformation	Sex			Total	Sex Ratio (Per cent. Male)	
	Male	Female	Not Determined			
Anencephalus ..	69	159	2	230	30·3	30·5
Anencephalus with Spina Bifida	29	64	3	96	31·2	
Spina Bifida ..	106	117	5	228	47·5	46·1
Spina Bifida with Hydrocephalus	69	91	—	160	43·1	
Encephalocele ..	19	19	—	38	50·0	
Hydrocephalus ..	83	68	1	152	55·0	
Total	375	518	11	904	42·0	

For anencephalus (including anencephalus with spina bifida) certain peculiarities of the sex ratio noted by McKeown and Lowe (1951) and MacMahon and McKeown (1952) are also found in the present series (Table III). Sex ratio is low in first births and at short gestations. The increasing sex ratio with increasing length of gestation is most marked in first births.

TABLE III
SEX RATIO OF ANENCEPHALUS

Gestation* (wks)	Birth Order					
	1		2 and over		Total	
	No.	Sex Ratio Percent. Male	No.	Sex Ratio Percent. Male	No.	Sex Ratio Percent. Male
Under 33 ..	41	19·5	28	35·7	69	26·1
33–37 ..	55	23·6	75	29·3	130	26·9
38 and over	46	37·0	60	36·7	106	36·8
Total ..	142	26·8	163	33·1	305	30·2

*Excluded are five cases of unknown sex, fifteen of unknown gestation, and one of unknown birth rank.

Duration of gestation is also generally shorter for first than for later births (Table IV). These differences are not statistically significant, but they are consistent with significant trends noted in the somewhat larger Birmingham material.

TABLE IV
PERCENTAGE DISTRIBUTION OF ANENCEPHALICS BY DURATION OF GESTATION

Birth Order	Gestation (wks)						Total	
	Under 33		33–37		38 and over			
	No.	%	No.	%	No.	%	No.	%
1	41	28·9	55	38·7	46	32·4	142	100
2 and over	28	17·2	.75	46·0	60	36·8	163	100

$\chi^2 = 5{\cdot}96$, $n = 2$, $p \simeq 0{\cdot}05$

MacMahon and McKeown (1952) suggested the possibility that the change in sex ratio with increasing length of gestation is related to greater severity or earlier onset of hydramnios in female anencephalics although, in their material, the total incidence of hydramnios was the same in the two sexes. The fact that the overall incidence of hydramnios is also higher in females in this series (Table V) points in the same direction. Table V also shows an increasing

TABLE V
PERCENTAGE INCIDENCE OF HYDRAMNIOS IN CASES OF ANENCEPHALUS

Birth Rank	Sex		Total
	Male	Female	
1	34·2	37·5	36·6
2	36·0	46·4	43·2
3	41·7	48·1	46·2
4+	29·4	65·4	51·2
Total ..	34·8	44·6	41·6

incidence of hydramnios with birth order, which suggests that hydramnios is not the explanation of the earlier onset of labour in firstborn anencephalics.

INCIDENCE RELATED TO COLOUR, RACE, AND RELIGION.—The great majority of births in Rhode Island are to white parents. According to central statistics, non-white births constitute only about two and a half per cent. of the total; the proportion of non-whites among hospital births is even lower, being only 1·98 per cent. in our comparison series. There are therefore too few non-whites in this series for adequate examination of incidence in whites and non-whites separately. We can however compare the number of non-whites in the comparison series with that in the malformation series.

Percentages of non-whites are given in Table VI; they are significantly fewer in the anencephalic group and more frequent in the hydrocephalic group than in the comparison series. The percentage is also low for spina bifida. If 1·98 per cent. is accepted as the true proportion of non-white births in these hospitals, the probability of finding no non-whites in 310 anencephalics by chance is $(0{\cdot}9802)^{310}$, or approximately 1 in 500.

TABLE VI
FREQUENCIES OF NON-WHITE BIRTHS

Diagnosis	Number* of Known Colour	Non-White		Difference from Comparison Series
		Number	Percentage	
Comparison Series	1,563	31	1·98	—
Anencephalus ..	310	0	0	−2·0±0·8†
Spina Bifida ..	401	4	1·0	−1·0±0·7
Hydrocephalus ..	140	7	5·0	+3·0±1·3†
All Malformations	851	11	1·29	−0·69±0·56

*Colour was not recorded for 53 (5·9 per cent.) of 904 malformations and 107 (6·4 per cent.) of 1,670 in the comparison series.
†Difference exceeds twice its standard error.

Several writers have suggested that American Negroes experience a low incidence of malformations as a group, but the evidence has not been convincing. A low incidence is suggested by centrally compiled birth and death certificate material (U.S. Public Health Service, 1949), but such material is undoubtedly subject to unequal ascertainment in the two groups. Other writers have demonstrated that deaths ascribed to malformation form a lower percentage of early deaths in Negroes than in whites (Clements, 1931; Murphy, 1936; Potter, 1940; Gruenwald, 1941), but this percentage is of course influenced by total still-birth and infant death rates, and is no indication of the relative frequencies of malformations in the total population of births. Murphy (1940) found the death rate from malformations to be 5·7 and 3·2 per thousand live births for whites and Negroes respectively, but the malformed infants were not derived solely from the general population on which the rates were based.

The suggestion in the present data that incidence of anencephalus and spina bifida is lower in non-whites than in whites, but that the incidence of hydrocephalus is higher offers further evidence that for race, as for other variables, examination of individual malformations is more profitable than consideration of malformations as a group.

The material in Table VII was collected to examine the relationship between religion and malformation, in view of known differences between religions in attitudes toward contraceptive practices and possibly to attempted abortion. No difference between Catholic and "Other" (almost entirely Protestant) religions is evident. However, somewhat surprisingly a low proportion of Jews is found in the malformation groups. This proportion is lowest for anencephalus and highest for hydrocephalus. If the percentage distribution of the comparison series is used to estimate the total number of Jewish births, the incidences of anencephalus, spina bifida, and hydrocephalus are 0·30, 0·60, and 0·30, respectively. These rates are equivalent to about one-seventh, one-quarter, and one-third of the corresponding figures for Gentiles. So far as we are aware, a low incidence

TABLE VII
PERCENTAGE DISTRIBUTIONS ACCORDING TO RELIGION

Religion*	Malformations								Comparison Series (*b*)		Difference (*a*)−(*b*)
	Anencephalus		Spina Bifida		Hydrocephalus		Total (*a*)				
	No.	%	No.	%	No.	%	No.	%	No.	%	
Catholic ..	227	70·28	310	73·99	104	68·42	641	71·70	1,123	69·28	+2·42±1·91
Jewish ..	2	0·62	4	0·95	2	1·32	8	0·89	64	3·95	−3·06±0·69†
Other	94	29·10	105	25·06	46	30·26	245	27·40	434	26·77	+0·63±1·85
Total	323	100	419	100	152	100	894	100	1,621	100	

*Religion was not stated for ten (1·1 per cent.) of 904 malformations and 49 (2·9 per cent.) of 1,670 comparison births.
†Difference exceeds twice its standard error.

of central nervous malformations in Jews has not been commented upon previously. Neither the low incidence in Jews nor that in non-whites can be accounted for by any unusual parity composition of these groups, since the differences in incidence are much larger than those between individual birth ranks.

RHESUS FACTOR AND ABO BLOOD GROUPING.—Distributions according to rhesus factor and blood group were examined in view of the possibility of their relevance to the different racial incidences noted above. A further indication for such an examination was the demonstration by Waterhouse and Hogben (1947) that matings between group O mothers and group A fathers produce a relative deficiency of group A children compared to other matings.

Results of blood examination were recorded more consistently, and for a longer period at the largest maternity hospital than at the four smaller institutions. The examination is therefore confined to propositi from this hospital, which contributed 480 (53·1 per cent.) of the malformations, and 868 (52·0 per cent.) of the comparison subjects. Blood group data were not available for the whole of the period surveyed, but were recorded for the mothers of 289 (60·2 per cent.) malformations and 542 (62·4 per cent.) comparison subjects. Rhesus status, but not ABO group, was available for an additional 31 mothers of malformations and for 37 in the comparison series.

The proportion of Rh-negative mothers is 11·7 per cent. (of 111) for anencephalus, 15·5 per cent. (of 148) for spina bifida, 11·5 per cent. (of 61) for hydrocephalus, and 13·4 per cent. (of 320) for all malformations combined, none of which percentages differs significantly from the corresponding value of 15·0 per cent. (of 579) in the comparison series.

In Table VIII the percentage distributions by ABO groups are compared, and again there are no significant differences between mothers of malformed and those of normal babies. We have examined these distributions for the period 1948–52 when information was available for 86·4 per cent. and 93·2 per cent. of the two groups, and, also, the percentage distributions by ABO group separately for Rh-positive and Rh-negative mothers. No noteworthy differences between malformations and comparison subjects were evident.

Wiener (1946) noted six families in which spina bifida and erythroblastosis had both appeared, and suggested that at least one type of spina bifida may be produced by Rh-sensitization. In the present material there were also at least six families in which a malformation was followed by the birth of an infant with erythrobalstosis. Record and McKeown (1950a) suggested that such families are the result of the fortuitous coincidence of two conditions neither of which is uncommon. Our data support this view, but the detection of sensitization effects occurring in a small proportion of families would require information on other members besides the mother. However, it does not seem likely that sensitization to the Rh or ABO groups is an aetiological factor in any appreciable proportion of malformations of the central nervous system.

SEASON OF BIRTH.—Table IX (opposite) shows incidence of malformations in 3-monthly periods. The standard quarters are used, since analysis by months did not suggest any more profitable grouping. That no seasonal variation in incidence of anencephalus was found is surprising in view of the report of McKeown and Record (1951), who noted among 158,307 births in Birmingham a significant excess of anencephalics born in the half-year October to March, although no seasonal change in incidence was evident for spina bifida or hydrocephalus. This high incidence of anencephalus in the winter months was independent of seasonal fluctuations in the proportions of births of various birth ranks. The large material on still births (based on 763,788 total births) published by the Registrar-General for Scotland showed similar relationships.

TABLE VIII

PERCENTAGE DISTRIBUTIONS BY BLOOD GROUP*

Blood Group	Malformations								Comparison Series (*b*)		Difference (*a*)−(*b*)
	Anencephalus		Spina Bifida		Hydrocephalus		Total (*a*)				
	No.	%	No.	%	No.	%	No.	%	No.	%	
O	51	49·0	64	48·5	25	47·2	140	48·44	242	44·65	+3·79±3·63
A	39	37·5	48	36·4	18	34·0	105	36·33	223	41·14	−4·81±3·56
B	9	8·7	16	12·1	8	15·1	33	11·42	62	11·44	−0·02±2·32
AB	5	4·8	4	3·0	2	3·8	11	3·81	15	2·77	+1·04±1·27
Total	104	100	132	100	53	100	289	100	542	100	

*This Table is based on malformations and comparison births in one hospital only. Blood group is unknown for 191 (39·8 per cent.) of 480 malformations and 326 (37·6 per cent.) of 868 comparison births.

TABLE IX

INCIDENCE OF MALFORMATIONS BY SEASON OF BIRTH

Quarter	Total Births	Malformations							
		Anencephalus		Spina Bifida		Hydrocephalus		Total	
		Number	Per mille	Number	Per mille	Number	Per mille	Number	Per mille
Jan.–March ..	40,638	77	1·89	106	2·61	34	0·84	217	5·34
April–June ..	41,387	79	1·91	101	2·44	36	0·87	216	5·22
July–Sept. ..	44,324	87	1·96	109	2·46	38	0·86	234	5·28
Oct.–Dec. ..	42,305	83	1·96	110	2·60	44	1·04	237	5·60
Total	168,654	326	1·93	426	2·53	152	0·90	904	5·36

We have examined our material from various aspects in an unsuccessful attempt to explain the absence of seasonal influence in the Rhode Island series. Restriction of the examination to propositi born in the period 1940–47 (the years covered by the Birmingham inquiry) was unfruitful. In Table X, percentage distributions by quarter-year are given for first-born anencephalics and comparison subjects, since the seasonal difference seemed to be most marked in first births in Birmingham. The percentage of anencephalics is high in the quarter October-December, but not significantly so, and the percentage in the first quarter is by contrast low. Examination according to month of last menstrual period also revealed no difference between anencephalics and normal births.

TABLE X

PERCENTAGE DISTRIBUTION BY QUARTER-YEARS OF FIRST-BORN ANENCEPHALICS AND COMPARISON SUBJECTS

Quarter	Anencephalics		Comparison Series		Difference
	No.	%	No.	%	
Jan.–March	33	22·0	171	24·5	$-2·5 \pm 3·8$
April–June	33	22·0	169	24·2	$-2·2 \pm 3·8$
July–Sept.	40	26·7	186	26·6	$+0·1 \pm 4·0$
Oct.–Dec.	44	29·3	173	24·7	$+4·6 \pm 3·9$
Total	150	100	699	100	

It would be unwise to conclude from this data that the seasonal variation in incidence of anencephalus noted in Birmingham and Scotland is not present in the United States, and that this is a major difference in the epidemiological behaviour of anencephalus in the two countries. We have elsewhere found that the selective features entailed in limitation of observations to hospital material may influence appreciably the magnitude of demonstrated associations (Ingalls and others, 1953), and it is possible that some such mechanism is operating in the present instance. Were the presence of such a difference in seasonal influence between the two countries confirmed, however, it would be of considerable interest.

INCIDENCE OF MALFORMATIONS OF THE CENTRAL NERVOUS SYSTEM IN SIBLINGS.—Since the mothers of the propositi were not interviewed in the present inquiry, our information on siblings is limited to:

(1) such data on previous siblings as was recorded in the obstetric case records;

(2) malformed siblings who were propositi by reason of birth in one or other of the study hospitals;

(3) all subsequent siblings, malformed and normal, born in Providence Lying-In Hospital after propositi born in that hospital.

This hospital maintains a unit record system which enables records to be readily inspected for all births of the same mother occurring in the hospital. Our estimate of risk to subsequent siblings is derived from this source. Fraternities containing two malformed members are listed in the Appendix to this paper.

The incidence of malformations of the central nervous system in siblings born subsequent to the first propositus in Providence Lying-In Hospital is shown in Table XI. 463 mothers gave birth to 480 propositi in this hospital and to one affected sibling who was not a propositus. The total incidence in subsequent siblings (5·3 per cent.) is rather higher than the estimate of 2·8 per cent. given by Record and McKeown (1950b). Penrose (1946) found seven (1·5 per cent.) affected in 471 siblings. Hindse-Nielsen (1938) found 28 (5·1 per cent.) affected among 548 sibs of patients with spina bifida.

TABLE XI

INCIDENCE OF MALFORMATIONS OF THE CENTRAL NERVOUS SYSTEM IN SIBLINGS BORN AFTER THE FIRST PROPOSITUS IN PROVIDENCE LYING-IN HOSPITAL

Type of First Malformation	Number of Fraternities	Number of Subsequent Siblings	Central Nervous Malformations	
			No.	%
Anencephalus	162	119	6	5·0
Spina Bifida	215	166	11	6·6
Hydrocephalus	86	54	1	1·9
Total	463	339	18	5·3

The number of sibs following hydrocephalic propositi in the present material is too small for calculation of rates, but the limited data agree with the observation of Penrose (1946) and of Record and McKeown (1950a) that recurrence of malformation is less frequent after hydrocephalus than after anencephalus or spina bifida. This is contrary to the material presented by Murphy (1940), but the discrepancy may be accounted for by the fact that in Murphy's data spina bifida with hydrocephalus appears to have been classified as hydrocephalus.

It is of both practical and theoretical interest to know the relative risk of recurrence of the same or different malformations. For this purpose we have combined our own material with that of Record and McKeown (1950a) in Table XII.

TABLE XII

RISK OF RECURRENCE OF THE SAME AND OF DIFFERENT MALFORMATIONS OF THE CENTRAL NERVOUS SYSTEM*

Diagnosis in First Propositus ..		Anencephalus	Spina Bifida	Hydrocephalus
Number of Subsequent Siblings ..		330	384	116
Number of Subsequent Siblings affected	Anencephalus	1	7	—
	Spina Bifida	5	12	1
	Hydrocephalus	1	—	1
Percentage Incidence of Subsequent Siblings affected	Anencephalus	1·2	1·8	—
	Spina Bifida	1·5	3·1	0·9
	Hydrocephalus	0·3	—	0·9
Percentage Incidence in all Births†		0·21	0·25	0·09

*The Table is based on a combination of the present data with that of Record and McKeown (1950a). Only malformations subsequent to the first propositus are included.
†Obtained by combining the estimates of the two series.

The examination is limited to the risk following the first propositus and even in this combined material the numbers are small. The Table compares the risk of each malformation in subsequent siblings with that in the general population, and the results indicate that:

(1) Following anencephalus, the risk of both anencephalus and spina bifida is considerably raised, being about six times that in the general population. The risks of anencephalus or spina bifida are approximately equal. Hydrocephalus in a subsequent sib was observed only once.

(2) Following spina bifida, there is a similar relationship, although spina bifida is somewhat more frequent in subsequent siblings than anencephalus; the risk of anencephalus is about seven times and that of spina bifida about twelve times greater than in the general population. Hydrocephalus was not observed in these fraternities.

(3) The number of subsequent sibs following hydrocephalus is small, but the limited data suggest that hydrocephalus is more common in subsequent siblings than anencephalus and/or spina bifida.

The tendency of anencephalus and spina bifida to recur or alternate in the same fraternity is confirmed by a wider examination of the literature. 74 fraternities in which two or more malformations of the central nervous system occurred may be assembled as follows:

35 from the present series (Appendices A and B);
26 from Record and McKeown (1950a, Appendix A, Groups I and II);
7 from Penrose (1946);
5 from Worcester, Stevenson, and Rice (1950);
1 from Böök and Rayner (1950).

These fraternities are unselected in so far as they are not derived from isolated reports of affected families. For reasons already given, the fraternities reported by Murphy (1940) are excluded.

In 24 of these fraternities in which the first malformation was anencephalus, subsequent malformations were anencephalus 12, spina bifida 10, hydrocephalus 2.

In 44 fraternities in which the first malformation was spina bifida, subsequent malformations were anencephalus 19, spina bifida 25, hydrocephalus 1.

Following hydrocephalus (six fraternities) anencephalus occurred twice, spina bifida once, and hydrocephalus three times.

The implication is clear that the factors responsible for the increased incidence in certain fraternities operate on anencephalus and spina bifida as one unit and hydrocephalus as another distinct entity.

OTHER MALFORMATIONS IN SIBLINGS.—The index cases (first propositi) had 307 previous and 321 subsequent siblings (total 628), excluding suspected or confirmed cases of central nervous malformation and twins of propositi; seven of these exhibited a variety of malformations other than those of the central nervous system, details of which are given in Appendix D. The incidence of other malformations in siblings (1·1 per cent.) is approximately the same as that of non-nervous system malformations in the general population.

DISCUSSION

Evidence is accumulating that anencephalus and spina bifida share common aetiological factors. McKeown and Record (1951) summarized certain common characteristics and other features of similarity have become apparent in the present inquiry:

(1) Females predominate in both, although more in anencephalus than in spina bifida;
(2) Both have shown a declining incidence in the post-war years in Birmingham and in Rhode Island;
(3) Both exhibit a low incidence in second births and a high incidence in first births and those of higher rank;

(4) In both, association with maternal age is relatively small compared to the association with birth order;

(5) Both show a low incidence in Jews as compared with Gentiles, and in American non-whites as compared with whites;

(6) Both recur or alternate in affected fraternities.

Seasonal variation in the incidence of anencephalus, but not in that of spina bifida, was observed in Birmingham and Scotland, but not in the present inquiry. Taken in conjunction with the inferred similarity in embryological development of anencephalus and spina bifida, this epidemiological behaviour suggests that it is reasonable for the present to regard these malformations as aetiologically related.

Hydrocephalus when not accompanying spina bifida, on the other hand, exhibits certain distinguishing features of behaviour:

(1) Greater frequency in males;

(2) Association with maternal age rather than with birth order;

(3) Higher incidence in non-whites;

(4) Tendency to repeat in affected fraternities;

(5) Comparative infrequency in fraternities containing members with anencephalus or spina bifida.

Hydrocephalus thus appears to be aetiologically distinct from the anencephalus-spina bifida group.

With the possible exception of the association with birth order and the decline in incidence in the post-war years, none of the associations yet demonstrated for anencephalus and spina bifida can be explained only in terms of either genetic or environmental factors. We have shown elsewhere that in Rhode Island the high incidence in first births as compared with later births was limited to the years of high incidence, 1940–47 (Ingalls and others, 1953). This is very suggestive of the influence of factors in the external environment of the mother. However, it cannot be determined whether these factors influence the incidence of the malformations or merely the proportion which are carried to term and diagnosed.

Explanations of either a genetic or environmental nature might be offered to explain the low incidence of these malformations in Negroes and in Jews. It is difficult to think of particular environments which are common to Negroes and Jews in the United States; it would perhaps be easier to attribute these differences to genetic influences, but it would be just as difficult to provide specific suggestions regarding the genetic mechanism involved.

Similarly, the high incidence in siblings as compared with the general population may be attributed to the genetic constitution of the parents or the continuation of particular environments within the fraternity. The observation of Record and McKeown (1950a) that the abortion rate in affected fraternities is especially high in the pregnancy immediately preceding the malformed individual is particularly interesting. If it were confirmed that the risk within the fraternity is localized to particular time periods, it would indicate that environmental factors were largely responsible for the high incidence of these malformations in siblings.

Since the operation of genetic or environmental factors has not been determined with certainty, it is hardly surprising that no specific genetic mechanism or environment has been ascertained. On the assumption that the excess of abortions in anencephalic fraternities is due to early anencephalus, Böök and Rayner (1950) concluded that the condition was the result of a lethal genotypical factor, possibly a simple recessive. A similar view was expressed by Polman (1950). Such statements are however purely speculative. The high mutation rate necessarily assumed in such an hypothesis is a major difficulty.

Nor can it be said that any limited environment is incriminated. Association with birth rank, secular change, and race could all indicate a variety of associated environmental factors.

Interpretation of the epidemiological data so far collected on hydrocephalus is difficult, since the nature of the material permits consideration of the malformation only as a single entity. Pathological differences between types of hydrocephalus and differences in age at onset make it certain that markedly different aetiological factors are responsible for individual types of the malformation. This may also of course be true for anencephalus and spina bifida, but it has not been shown to be so, and there is no choice but to regard them, for the present, as being of uniform aetiology. However where such differences are known to be present, as in hydrocephalus, examination of material which takes account of them is likely to be most profitable. It seems unlikely therefore that any considerable advance in knowledge of this malformation will be made by statistical or genetic investigation without coincident pathological examination of the material.

Summary

From the obstetric case records of the five largest maternity units in the State of Rhode Island, information was abstracted on the mothers of 904 infants with anencephalus, spina bifida, hydrocephalus, or encephalocele, and those of 1,670 infants selected at random (comparison series). Births in these

hospitals accounted for over 67 per cent. of those in the whole state, and are believed to be representative of all births occurring in the central and northern sections of the state.

Incidences of anencephalus, spina bifida, and hydrocephalus were 1·93, 2·53, and 0·90 per thousand total births respectively. The incidence of all three malformations was highest in the war years and declined during the post-war period. Certain associations of the sex ratio of anencephalus are recorded.

The percentage of non-whites was lower for anencephalus and spina bifida than for the comparison series (significantly so for anencephalus), and for hydrocephalus it was significantly higher. No difference in incidence of malformations was evident among the progeny of Catholic and Protestant mothers, but significantly fewer malformations than expected were found among Jews. Incidences of the three malformations in Jews were one-seventh, one quarter, and one-third of the corresponding rates for Gentiles. No differences between mothers of malformed infants and those in the comparison series were noted in respect of percentage distribution by Rh factor or ABO blood group. No significant seasonal variations in incidence were found.

The incidence of malformations of the central nervous system in siblings born subsequent to a propositus was 5·3 per cent., the risk being highest after spina bifida and lowest after hydrocephalus. Anencephalus and spina bifida appear to alternate freely in fraternities in which one such malformation has occurred. Hydrocephalus is relatively infrequent after anencephalus or spina bifida, but is the most common malformation following a hydrocephalic propositus.

Seven of 628 siblings of propositi (1·1 per cent.) had malformations other than those here considered. This incidence is approximately the same as that of non-nervous system malformations in the general population.

Reasons are given for the belief that anencephalus and spina bifida are closely related aetiologically as well as embryologically, whereas hydrocephalus appears to be aetiologically distinct.

We are indebted to the administrative and obstetric staffs of the Providence Lying-In, Roger Williams General, St. Josephs, Pawtucket Memorial, and Woonsocket General Hospitals, who kindly gave access to their records.

We are particularly grateful for the encouragement of Miss Gertrude Cahir and Drs Harmon P. Jordon, Alfred L. Potter, Walter S. Jones, and John G. Walsh of the Providence Lying-In Hospital, where the study was begun.

REFERENCES

Böök, J. A., and Rayner, S. (1950). *Amer. J. hum. Genet.*, **2**, 61.
Clements, F. (1931). *Hum. Biol.*, **3**, 397.
Gruenwald, P. (1941). *Illinois med. J.*, **79**, 55.
Hindse-Nielsen, S. (1938). *Acta chir. scand.*, **80**, 525.
Ingalls, T. H., Pugh, T. F., and MacMahon, B. (1953). (In press).
McKeown, T., and Lowe, C. R. (1951). *Hum. Biol.*, **23**, 41.
—— and Record, R. G. (1951). *Lancet*, **1**, 192.
MacMahon, B., and McKeown, T. (1952). *British Journal of Social Medicine*, **6**, 265.
——, Record, R. G., and McKeown, T. (1951). *Ibid.*, **5**, 254.
Murphy, D. P. (1936). *South med. J., Bgham, Ala.*, **29**, 79.
—— (1940). "Congenital Malformations", University of Pennsylvania Press, Philadelphia (1947). 2nd ed., Lippincott, Philadelphia.
Penrose, L. S. (1946). *Ann. Eugen. Camb.*, **13**, 73.
Polman, A. (1950). *Genetica*, **25**, 29.
Potter, E. L. (1940). *J. Amer. med. Ass.*, **115**, 996.
Record, R. G., and McKeown, T. (1949). *British Journal of Social Medicine*, **3**, 183.
—— —— (1950a). *Ibid.*, **4**, 26.
—— —— (1950b). *Ibid.*, **4**, 217.
—— —— (1951). *Ann. Eugen. Camb.*, **15**, 285.
U.S. Public Health Service (1949). Vital Statistics Special Reports, **36**, 216.
Waterhouse, J. A. H., and Hogben, L. (1947). *British Journal of Social Medicine*, **1**, 1.
Wiener, A. S. (1946). *Amer. J. clin. Path.*, **16**, 319.
Worcester, J., Stevenson, S. S., and Rice, R. G. (1950). *Pediatrics*, **6**, 208.

APPENDIX

APPENDIX A

FRATERNITIES CONTAINING AN INDEX PROPOSITUS AND A SUBSEQUENT PROPOSITUS OR AFFECTED SIBLING, BOTH BORN IN PROVIDENCE LYING-IN HOSPITAL

Reference Number	Siblings in Order of Birth											
	1	2	3	4	5	6	7	8	9	10	11	12
424	*M*	*M*	*M*	*M*	M.1*	M.3*	*M*					
425	(?)	(?)	(?)	(?)	(?)	(?)	(?)	(?)	(?)	*M*	†M.1*	M.2*
426	F.1*	F.3*										
427	F.1*	*M*	F.1*	*M*								
428	M.2*	*M*	M.6*									
429	F.2*	‡	‡	M.3*								
430	†F.3*	F.3*										
431	*M*	*M*	F.3*	*M*	F.1*	*M*	*M*					
432	M.4*	M.4*										
433	(*M*)	M.4*	M.4*	‡								
434	*M*	*M*	*M*	M.4*	M.1*							
435	F.4*	M.1*										
436	F.4*	*M*	F.3*	*F*								
437	*M*	F.4*	*M*	M.4*								
438	(?.1)	(*F*)	‡	†F.4*	F.1*							
439	*M*	F.5*	*F*	F.1								
440	(*F*)	(*F*)	(*M*)	F.5*	*M*	†? 3*						
441	M.6*	M.6*	†M									

KEY

(1) Sex is shown by M, F, ?.

(2) Malformed individuals are in bold type, normal in italics.

(3) The diagnosis of defects of the central nervous system is indicated by numerals as follows:

1. Anencephalus
2. Anencephalus with Spina Bifida
3. Spina Bifida
4. Spina Bifida with Hydrocephalus
5. Encephalocele
6. Hydrocephalus.

(4) Other diagnoses are given in the notes at the end (†).

(5) * propositus.

(6) ‡ miscarriage.

(7) Individuals not born in Providence Lying-In Hospital are shown in brackets.

APPENDIX B

OTHER FRATERNITIES CONTAINING TWO AFFECTED MEMBERS BORN IN STUDY HOSPITALS

Reference Number	Siblings in order of Birth							
	1	2	3	4	5	6	7	8
442	*(F)*	*(M)*	M.3	M.4*				
443	F.4	*M*	*F*	†F.1*	*M*			
444	‡	*F*	‡	†(M.2)*	M.1*			
445	*(?)*	(M.3)*	*F*	*M*	M.1*	*F*		
446	*F*	(M.2)*	F.4*					
447	*(M)*	(F.2)*	F.4*					
464	*(?)*	(F.1)*	(F.4)*					
465	*(M)*	(F.4)*	(F.2)*					
466	(M.3)*	*(?)*	*(?)*	*(?)*	(F.1)*			
467	(M.3)*	(F.3)*						
468	(F.3)*	*(F)*	(F.3)*					
469	*(M)*	(F.5)*	(F.3)*	‡				
470	*(?)*	(M.4)*	(F.4)*					
471	*(?)*	*(?)*	*(M)*	(M.2)*	‡	(F.3)*		
472	*(?)*	*(?)*	*(?)*	(F.2)*	*(?)*	*(?)*	*(?)*	(F.2)*
473	‡	(F.2)*	(F.1)*					
474	*(M)*	(F.1)*	*(M)*	(F.1)*	*(F)*	‡	*(M)*	

APPENDIX C

FRATERNITIES CONTAINING A PROPOSITUS FROM P.L.I. HOSPITAL AND A PREVIOUSLY MALFORMED SIBLING NOT BORN IN A STUDY HOSPITAL (DIAGNOSIS THEREFORE UNCONFIRMED)

Reference Number	Siblings in Order of Birth										
	1	2	3	4	5	6	7	8	9	10	11
448	*M*	(? 6)	*(M)*	*(M)*	(M.3)	F.1*					
449	(? 3)	‡	F.1*								
450	*(F)*	(F.6)	*(F)*	*(M)*	*(M)*	M.3*					
451	(F.3)	(M.3)	‡	‡	*(M)*	†(F)	‡	*(F)*	†(?)	(? 3)	M.4*
452	(M.3)	†M.4*									
453	*F*	*(F)*	(?.1)	F.6*	*F*	*F*	*F*	*M*			

APPENDIX D

FRATERNITIES CONTAINING A PROPOSITUS FROM P.L.I. HOSPITAL AND A SIBLING WITH A MALFORMATION OTHER THAN ONE OF THE CENTRAL NERVOUS SYSTEM

Reference Number	Siblings in Order of Birth				
	1	2	3	4	5
454	F.3*	*M*	*M*	†M	
455	†?	†F.3*	*M*	*F*	
456	†M	M.3*			
457	*M*	F.4*	†M		
458	†(?)	†M.4*			
459	†M	M.1*	*F*	*M*	*M*
460	†M	F.1*	*F*	*M*	*M*
463	*M*	†M	F.1* *F*		

Reference Number of Fraternity	Birth Rank	(†)Diagnostic Notes
425	11	Propositus; rudimentary external genitalia
430	1	Propositus; harelip
438	4	Propositus; omphalocele
440	6	Propositus; microcephalus; large umbilical hernia
441	3	Suspected congenital heart disease
444	4	Also extrophy of the heart
451	6,9	Congenital anomaly, unspecified
452	2	Propositus; oxycephaly
454	4	Right talipes equino-varus
455	1	Omphalocele
	2	Propositus; extrophy of bladder; primitive cloaca
456	1	Enlarged heart; ? congenital cystic kidney
457	3	Congenital duodenal stenosis
458	1	"Abnormal infant"
	2	Propositus; enlarged liver and spleen
459	1	Tumour in neck
460	1	Scaphocephaly
463	2	Spinal deformity

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ABO BLOOD GROUPS AND HUMAN FERTILITY

BY

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The fact that those who lack the blood group antigen A have anti-A antibody, and those who lack B have anti-B (Landsteiner, 1900, 1901), leads in a number of cases to an incompatibility between maternal serum and foetal antigens inherited from the father. Such serologically incompatible pregnancies have been thought to be a possible cause of various morbid conditions in foetus or mother (*e.g.*, Gruhzit, 1923, 1924), although Tovey (1945) considers that placental resistance to the passage of agglutinin, and the presence of ABO substance in foetal plasma, together with the low sensitivity of foetal red cells, may greatly lessen the effect on the foetus.

Hirszfeld and Zborowski (1925, 1926) found that, in the mating class Father A × Mother O, there were relatively fewer A offspring than in the reciprocal class Father O × Mother A, and also that there were relatively fewer O children from Mother A × Father O than from Mother O × Father A. Later results of Hirszfeld (1928) agreed with this, but Koller (1931) showed that the findings were not significant. An exhaustive review of the literature by Hirszfeld (1934) reversed his previous conclusions, and his further work (1938) did not confirm them. Levine (1943) assembled data apparently in support not of Hirszfeld's theory of constitutional incompatibility between a mother and a foetus of different groups but of Gruhzit's theory of serological incompatibility.

Waterhouse and Hogben (1947) analysed twelve carefully chosen family studies made between 1927 and 1944 primarily to decide between the modes of inheritance of the ABO blood groups proposed by von Dungern and Hirszfeld (1910) and Bernstein (1924, 1925). Selecting the largest reciprocal mating class—namely Father A × Mother O and Father O × Mother A—in the pooled material from these studies, they found:

(*i*) a significant shortage of families of the class Father A × Mother O as compared with its reciprocal;

(*ii*) a highly significant shortage of group A children from the class Father A × Mother O:

(*iii*) a sharp fall with increasing birth rank in the ratio of A to O children in the mating class Father A × Mother O.

They reckoned the loss at 25 per cent. of the group A children expected from the mating class Father A × Mother O, and suggested that the loss was due to early abortion.

As a result of the findings of Allan (1952a, b) of a significant excess of group B women in samples of cases of both abortion and sterility, Waterhouse and Hogben's collected material has here been analysed in terms of relative fertility by mating class. In this context the term *fertility* has been used synonymously with mean number of children per family, to avoid inconvenient periphrasis. No confusion with any more precise definition of the term is intended.

Results

Hirszfeld and Zborowski (1925) coined the term "homospecific" for a pregnancy in which the mother and foetus are of the same ABO group, and "heterospecific" for one in which their ABO groups differ. In this paper the term "heterospecific" is used instead for a pregnancy in which the red cells of the foetus are not only of a different ABO group from those of the mother but would also be agglutinated by the mother's serum *in vitro*. (The term "incompatible", unqualified, begs the question when applied to reproduction.) Similarly, a mating is here called heterospecific in which the wife has specific antibodies to one or both of the antigens possessed by her husband (*i.e.*, an incompatibility between the husband as donor and the wife as recipient). It will be seen that no class of homospecific mating can have heterospecific offspring (in Levine's sense), but that (most A and B individuals being heterozygous, *i.e. AO* or *BO*) all classes of heterospecific

mating except AB × O can have homospecific offspring. The father's group is put first, and the fathers in (for example) the mating classes B × B and B × AB are collectively termed "homospecific B fathers", while the fathers in the mating classes B × A and B × O are collectively termed "heterospecific B fathers".

The expected numbers of children in the sixteen mating classes in the Waterhouse-Hogben sample have been calculated by multiplying the expected number of families in each of the classes by 3·34, this being the overall average number of children per family in the 1,239 families in the sample. A homospecific mating is found in 793 of the families, these having an average of 3·34 children, while a heterospecific mating is found in 446 of the families, these having an average of 3·33 children. In 440 of the 793 homospecific families the father and mother are of the same group, and have an average of 3·33 children per family. The average number of children in the 840 families, homospecific and heterospecific, in which one or both parents are O is 3·37; in the 273 families with one or both parents B it is 3·34; in the 812 families with one or both parents A it is 3·33; and in the 113 families with one or both parents AB it is 3·29. Table I gives the nine homospecific mating classes in descending order of average numbers of children per family, and shows that, of the five classes of heterospecific mating with a homospecific reciprocal, three are more fertile than the reciprocal. When, however, the heterospecific mating classes are studied alone, the no less interesting fact appears that the A mothers are at the upper end of the table, the B mothers at the lower end, and the O mothers in the middle; and one is thereby led to consider the rest of the sample from this unexpected point of view.

TABLE I

ORDER OF FERTILITY OF ABO MATING CLASSES

No. Observed		Homospecific Mating Classes (Father first)	Average No. of Children per Family		Heterospecific Mating Classes (Father first)	No. Observed	
Families	Children					Children	Families
8	30	B×AB	3·75	—	—	—	—
17	62	B×B	3·65	4·00	AB×A	80	20
225	779	O×O	3·46	3·56	B×A	210	59
28	96	A×AB	3·43	3·47	B×O	229	66
244	835	O×A	3·42	3·28	A×O	686	209
189	599	A×A	3·17	3·13	AB×O	72	23
54	171	O×B	3·17	3·11	A×B	196	63
19	56	O×AB	2·95	2·33	AB×B	14	6
9	24	AB×AB	2·67	—	—	—	—

For this purpose, it is helpful to combine those mating classes in which the father or the mother is of any one group and specificity—*e.g.* B × B and B × AB—and when this is done the descending order of fertility of homospecific fathers is found to be B-O-A. The picture grows still clearer when the A and B parents of both specificities are taken alone, as in Table II.

TABLE II

ORDER OF FERTILITY OF A AND B PARENTS

Homospecific Parents			Heterospecific Parents		
Ratio of No. of Families Observed to No. Expected	Group of Parent	Average No. of Children per Family	Group of Parent	Ratio of No. of Families Observed to No. Expected	
1·04	B Father	3·68	3·67	A Mother	0·95
1·04	A Mother	3·31	3·51	B Father	1·04
0·90	B Mother	3·28	3·24	A Father	1·00
0·95	A Father	3·20	3·04	B Mother	1·05

If now the B parents of both sexes and both specificities are placed in descending order of fertility, as in Table III, and the A parents are placed alongside, the second pattern of reciprocity stems only partly from the first.

TABLE III

ORDER OF FERTILITY OF A AND B PARENTS

Group B Parents				Group A Parents		
Children				Children		
Ratio of No. Observed to No. Expected	Range from Observed to Expected	Average No. per Family	Specificity of Parent	Average No. per Family	Range from Observed to Expected	Ratio of No. Observed to No. Expected
1·15	+12	3·68	Homospecific Father	3·20	−66	0·91
1·09	+37	3·51	Heterospecific Father	3·24	−29	0·97
0·89	−30	3·28	Homospecific Mother	3·31	+39	1·03
0·96	−9	3·04	Heterospecific Mother	3·67	+13	1·05

On the other hand, when the AB and O parents of both sexes and both specificities are placed in descending order of fertility, as in Table IV (overleaf), and the O parents are placed alongside, the outcome is parallelism. When the homospecific O and AB parents are placed in descending order, as in Table V (overleaf), alongside the heterospecific O and AB parents, the result is again reciprocity.

TABLE IV

ORDER OF FERTILITY OF AB AND O PARENTS

Group AB Parents				Group O Parents		
Children				Children		
Ratio of No. Observed to No. Expected	Range from Observed to Expected	Average No. per Family	Specificity of Parent	Average No. per Family	Range from Observed to Expected	Ratio of No. Observed to No. Expected
0·86	−27	3·39	Heterospecific Father	—	—	—
1·00	+1	3·22	Homospecific Mother	3·46	+10	1·01
1·93	+12	2·67	Homospecific Father	3·40	+63	1·04
—	—	—	Heterospecific Mother	3·31	−23	0·98

TABLE V

ORDER OF FERTILITY OF AB AND O PARENTS

Homospecific Parents			Heterospecific Parents		
Ratio of No. of Families Observed to No. Expected	Group of Parent	Average No. of Children per Family	Group of Parent	Ratio of No. of Families Observed to No. Expected
0·98	O Mother	3·46	—	—	—
1·02	O Father	3·40	3·39	AB Father	0·85
1·04	AB Mother	3·22	3·31	O Mother	0·99
2·42	AB Father	2·67	—	—	—

These findings strongly suggest that, judging by the Waterhouse-Hogben sample, heterospecificity does not, of itself, lower fertility, and one or two further points may reinforce the argument. For example, 84 per cent. of the AB fathers in the sample, 83 per cent. of the B fathers, 56 per cent. of the A fathers, and none of the O fathers, are heterospecific, and—presuming A and B offspring to be equally, or almost equally, sensitive—this would lead one to expect the descending order of fertility of all fathers in the sample to be O-A-B-AB, but in fact it is B-O-AB-A. Similarly, 57 per cent. of the O mothers in the sample, 49 per cent. of the B mothers, 15 per cent. of the A mothers, and none of the AB mothers are heterospecific, and this would lead one to expect the descending order of fertility of mothers in the sample to be AB-A-B-O, whereas in fact it is O-A-AB-B. It is certainly possible that, if heterospecific parents did lose offspring, they would make up the loss by more pregnancies, as do parents who lose (full-term) offspring from Rh-incompatibility (Glass, 1949), or from hereditary acholuric jaundice (Race, 1942); and it is probable that, since all types of heterospecific mating except AB × O can give rise to homospecific offspring, many of these extra offspring would live. But if this did happen the chances would be small that heterospecific matings would have, as in the present sample, so nearly the same overall average number of children per family as homospecific parents; and there would still remain the question why heterospecific A fathers and B mothers were unable to make up the loss while heterospecific B fathers and A mothers more than made it up.

If, then, heterospecificity has no effect on fertility, some other reason must be sought for the curious differences of fertility found in the sample. Now Table VI shows that four of the eight mating classes with a higher than average number of children per family have a group B father, and also that all four classes with a B father are more fertile than any of the four classes with an A father—an event which would occur by chance 1 in 70 times (Fisher, 1951). It can also be seen that, in seven of the eight types of parent, some mating classes show an excess, and some a shortage, either of families or of children; whereas all four of the mating classes with a B father show an excess both of families and of children. Thus, to judge by this one sample, the B father, as such, is the most fertile type of parent among Caucasians, and may be provisionally regarded as a norm by whom all other types of parent may be judged, the descending order of fertility of all types of parent being shown in Table VII. The B father is also the type of parent with the

TABLE VI

ORDER OF FERTILITY OF ABO MATING CLASSES

Ratio of No. Observed to No. Expected		Major Mating Classes (Father first)	Average No. of Children per Family		Minor Mating Classes (Father first)	Ratio of No. Observed to No. Expected	
Families	Children					Children	Families
1·01	1·08	B×A	3·56	4·00	AB×A	0·97	0·81
1·06	1·10	B×O	3·47	3·75	B×AB	1·23	1·10
0·98	1·01	O×O	3·46	3·65	B×B	1·11	1·02
1·14	1·17	O×A	3·42	3·43	A×AB	1·17	1·14
0·97	0·96	A×O	3·28	3·13	AB×O	0·84	0·89
0·93	0·88	A×A	3·17	2·95	O×AB	0·65	0·74
0·87	0·82	O×B	3·17	2·67	AB×AB	1·93	2·42
1·08	1·01	A×B	3·11	2·33	AB×B	0·57	0·82

smallest *range* of fertility in the sample—from 3·75 for B × AB to 3·47 for B × O—the ascending order of width of range being B father, A father, O mother, O father, A mother, B mother.

TABLE VII

DESCENDING ORDER OF FERTILITY OF PARENTS (INCLUDING MATING CLASSES B × B, O × O, A × A, AND AB × AB)

Families			Group of Parent	Children		
No. Observed	Range from Observed to Expected	Ratio of No. Observed to No. Expected		Ratio of No. Observed to No. Expected	Range from Observed to Expected	No. Observed
150	+6	1·04	B Father	1·10	+49	531
542	+9	1·02	O Father	1·04	+62	1,841
523	−10	0·98	O Mother	0·99	−13	1,766
512	+12	1·02	A Mother	1·03	+52	1,724
58	−3	0·94	AB Father	0·93	−16	190
489	−11	0·98	A Father	0·94	−95	1,577
64	+3	1·04	AB Mother	1·00	+1	206
140	−4	0·97	B Mother	0·92	−39	443

When the eight types of parent are separated according to sex (Table VIII), it is seen that, while the descending order of fathers is B-O-A or B-AB-A, the order of mothers, though A-AB-B, is not A-O-B but O-A-B. Now, in this context, the fact that O mothers are less fertile than B fathers would suggest that the non-reciprocity of mothers with the B-O-A order of fathers is due not to O mothers but to A mothers; and that this is indeed so is clear from Table VI. Here it is seen that all four types of B father are more fertile than three of the four types of B mother, the exception being the mating class B × B, and that all four types of A father are less fertile than three of the four types of A mother, the exception being the populous class A × A. In other words, it is clear that the classes B × B and A × A, which could (other things being equal) have resembled the mother's side or neither side in respect of fertility, decidedly resemble the father's side, and that this distorts the sample as a whole. This effect can be eliminated—relatively, at least—by leaving these classes out of the sample along with AB × AB and O × O; when this is done there emerges the almost perfect reciprocity of Table IX. Given this order of fertility of fathers, its reciprocal, like its parallel, would occur in mothers by chance once in 24 times.

TABLE VIII

ORDER OF FERTILITY OF FATHERS AND MOTHERS (INCLUDING MATING CLASSES B × B, O × O, A × A, AND AB × AB)

No. Expected		Group of Father	Average No. of Children per Family		Group of Mother	No. Expected	
Families	Children					Children	Families
144	482	B	3·54	3·38	O	1,779	533
533	1,779	O	3·40	3·37	A	1,672	500
61	205	AB	3·28	3·22	AB	205	61
500	1,672	A	3·22	3·16	B	482	144

TABLE IX

ORDER OF FERTILITY OF FATHERS AND MOTHERS (EXCLUDING MATING CLASSES B × B, O × O, A × A, AND AB × AB)

No. Expected		Group of Father	Average No. of Children per Family		Group of Mother	No. Expected	
Families	Children					Children	Families
128	426	B	3·53	3·48	A	993	297
58	193	AB	3·39	3·31	O	1,010	302
302	1,010	O	3·35	3·31	AB	193	58
297	993	A	3·26	3·10	B	426	128

Moreover, this notable pattern leads, though not of necessity, to the even more striking pattern of Table X, where the descending order of blood groups of the four types of parent of more than average fertility is reversed in the parents of less than average fertility. This type of pattern might be thought to be due to the blood-group incidence,

TABLE X

DESCENDING ORDER OF FERTILITY OF PARENTS (EXCLUDING MATING CLASSES B × B, O × O, A × A, AND AB × AB)

Families			Group of Parent	Children		
No. Observed	Range from Observed to Expected	Ratio of No. Observed to No. Expected		Ratio of No. Observed to No. Expected	Range from Observed to Expected	No. Observed
133	+5	1·04	B Father	1·10	+43	469
323	+26	1·09	A Mother	1·13	+132	1,125
49	−9	0·85	AB Father	0·86	−27	166
317	+15	1·05	O Father	1·05	+52	1,062
298	−4	0·99	O Mother	0·98	−23	987
55	−3	0·95	AB Mother	0·94	−11	182
300	+3	1·01	A Father	0·99	−15	978
123	−5	0·96	B Mother	0·89	−45	381

but if that were really the cause, in whole or in part, the smallest samples—those of the AB parents—would be at the ends instead of very near the mean, and would, along with A and B, have a much more even sex distribution. It seems more likely that this pattern, together with that of Table X, gives some provisional support to the theory of Ford (1948) that the ABO blood groups are a system of balanced polymorphism, like sex—all the more so as such a system is believed to be invariably the outcome of a balance of opposed selective agencies, as, *e.g.*, maleness and femaleness. (It has been shown by Fisher (1930) that genes of *neutral* survival value must be rare). But that the ratio of the ABO genes may not, in fact, be stable is suggested by the high fertility of B × B and the low fertility of A × A, especially as the difference between these two classes is even greater in the much larger sample of Hirszfeld (1934), as analysed by Allan (1953).

Another process, however, by which an ABO balance might be maintained can be discerned in Table XI, from which the balance is seen to be largely due to a balance of reciprocal classes of mating. In this connection it is relevant to note that, just as in the mating class A × O the ratio of A/O children falls with rising birth-rank, so in the mating class O × A, if the children above the fifth birth-rank are ignored, rising birth-rank gives a steady fall in the ratio of O/A children. When he saw this in August, 1951, the writer accepted the suggestion put to him six months before by a colleague, Dr. William Hamilton, that the data point to direct genetic action, but this two-fold decline also hints at some environmental factor.

TABLE XI

COLLECTIVE FERTILITY OF COMPARABLE ABO MATING CLASSES

No. Expected		Classes of over Average Fertility (Father first)	Combined Observed Average No. of Children per Family	Classes of under Average Fertility (Father first)	No. Expected	
Families	Children				Children	Families
214·36	715·96	O×A	3·36	A×O	715·96	214·36
24·60	82·16	AB×A		AB×O	86·21	25·81
24·60	82·16	A×AB	3·35	O×AB	86·21	25·81
7·29	24·35	B×AB		AB×B	24·35	7·29
62·11	207·45	B×O	3·33	O×B	207·45	62·11
230·24	769·00	O×O	3·33	A×A	678·85	203·25
58·27	194·62	B×A	3·33	A×B	194·62	58·27
16·67	55·68	B×B	3·31	AB×AB	12·42	3·72

Hogben (1939) has said:

> Several things point to the importance of studying the influence of the uterine environment upon the characteristics of individuals. One is the high incidence of certain conditions amongst first-born children. Another is the high incidence of various malformations among women approaching the end of the child-bearing period.

And Ford (1948), writing of the effect of maternal age upon the penetrance of the genes in the offspring, has said:

> It is thought that a number of conditions, some of them in part genetic, are expressed with greater frequency in children as maternal age advances.

DISCUSSION

It has been shown that, in the present sample, all four types of A father are less fertile than all four types of B father. Now, the four types of A father—A × AB, A × B, A × O, and A × A—can have, together, offspring of all four ABO groups, *viz.*:

(*i*) B offspring, whose B gene is always inherited from the mother;

(*ii*) O offspring, with an O gene from the mother and another from the father;

(*iii*) AB offspring, with a B gene from the mother and an A gene from the father;

(*iv*) A offspring, all of whom, in the classes A × B and A × O, get their A gene from their father, and a large majority of whom, in the classes A × A and A × AB, get an A gene from their father.

But the only type of offspring common to these four classes are A offspring. Indeed A offspring and A fathers are the only relevant characters shared by these classes, suggesting that one or other or both of these characters must be the cause of their sub-mean fertility. This idea is made the more plausible by Waterhouse and Hogben's demonstration that the loss of offspring in the mating class A × O is a loss of A, not of O, offspring, and by the fact that a large majority of the offspring of the mating class A × A are also A. If, however, high mortality of A offspring, as such, were a basic cause of sub-mean fertility, the mating classes AB × A, B × A, and O × A would also be short of A children; but Waterhouse and Hogben have shown that there is no such shortage—relative, at any rate, to O children—in the mating class O × A, which is the largest, and the least fertile, of the three—no relative shortage, in other words, of A children whose A gene has come from their mother. This would suggest that the basic cause of the sub-mean fertility of A fathers is the fact that they, themselves and not their children or their wives, are A. If this were found to be the truth, it would be interesting to see whether men with two A genes were less fertile than those with one.

In the present sample, also, if the mating class B × B is left out, all three types of B mother are less

fertile than all three types of A mother (and all three types of B father). In the same way then, as with A, and in spite of the fact that the shortage of children in the mating class O × B is, as Waterhouse and Hogben have pointed out, a shortage of B children, one is led to think that the basic cause of the sub-mean fertility of B mothers, other than those with a B husband, is the fact that they themselves, and not their children or their husbands, are B. As with A fathers, it would be interesting to see whether B women with two B genes are less fertile than those with one.

Genetic action by *both* the parents in the mating class A × B might explain why that class is even less fertile than the classes O × B and A × O. On the other hand, the effect on AB offspring might be modified by the union of an A and B gene in these offspring. This would tally with the fact that AB parents are near the middle of the scale, if the class AB × AB is set aside, though it is notable that there is then a shortage of families with AB mothers. It is possible, too, that the parallelism shown in Table IV between O and AB parents, unlike the reciprocity of A and B parents shown in Table III, might be due to the fact that the genotype *AB* is doubly dominant, and *OO* recessive, while in most of the world A and B individuals are mainly heterozygous (*AO* or *BO*), not homozygous (*AA* or *BB*).

That the group O fathers and mothers are in the centre of the scale, at least when O × O is omitted (Table IX) might be expected from the partial reciprocity of the classes A × O and O × A, B × O and O × B, AB × O and O × AB, but the fact that the class O × O has a shortage of families, and a lower average number of children per family than any class with a group B father, hints at selection against the O gene. More support for this hypothesis can be got from classes A × O and O × A, apart from the fact that all the A offspring of these two classes have at least one O gene, and by virtue of the fact that A × O and O × A are only partially reciprocal. Thus if the 114 A × O families in which the father is known to be heterozygous are joined with the 135 families in which the mother is known to be heterozygous, the four kinds of mother-child combination give the following average numbers* of children per family:

(*i*) 2·23 for O ex O;
(*ii*) 2·06 for A ex A;
(*iii*) 2·01 for O ex A;
(*iv*) 1·83 for A ex O.

Of the remaining 95 A × O and 109 O × A families, there is a small number whose children are all of group O, and in these the average number of children is 0·49 for O ex O and 0·44 for O ex A. Thus, in both types of A × O and O × A family, there are fewer O children from A mothers than from O mothers. On the other hand, the average number of O children per family is 1·59 for both the 66 B × O matings and the 54 O × B matings.

If there were, in fact, selection either for or against any of the O, A, and B genes, the first question that would arise concerns the form such selection might take—the stage or stages, from conception onward, in the life-history of the offspring, at which selection might occur or be most severe. Other questions would concern the influence, if any, that might be exerted by other genes, such as the blood-group genes for Secretion, Lewis, and Rhesus; the possibility of differential selection against the sub-groups of A or the still obscure sub-groups of B; and the possibility that certain ABO mating classes are more prone than others to give rise to twins or triplets. In the meantime, however, whether there is, in fact, selection against the O, A, and B genes, or whether, on the contrary, the pattern of fertility found in this sample is due to chance, is a question which only further investigation can answer. The Waterhouse-Hogben sample was taken from several scattered Caucasian sources, and as the authors pointed out, is not entirely homogenous. This being so, the ideal source of a large enough sample to test the hypothesis would, at first sight, be a city, but few large cities, even in Britain, have homogeneous populations. Nor would the environs of cities be in very much better case, for the detailed surveys of Northern England by Roberts (1951), and of Wales and the Marches by Mourant and Watkin (1952), have uncovered strikingly different ABO blood-group distributions even in neighbouring valleys and villages.

Firmer evidence could be claimed if populations whose ABO blood-group distribution was very different from the Caucasian showed a pattern of fertility—given samples with roughly the same average family size—which essentially fitted the hypothesis. For example, in populations with a much higher A-gene frequency and a much lower B-gene frequency (*i.e.* in populations with a higher proportion of homozygous A husbands and wives, and a lower proportion of homozygous B husbands and wives) one might find that A, not B, parents were at the extremes; but there are very few such peoples. Indeed the frequency of the A-gene falls fairly steadily, while that of the B-gene rises, from Western Europe to Eastern Asia (Hirszfeld and Hirszfeld,

* The overall average for these 135 O × A families is 4·07, and for the 114 A × O families 4·06.

1919); and thus, as the Waterhouse-Hogben sample is composed of European and White American individuals, one would expect, other things being equal, to find its pattern of fertility maintained, and the range from B father to B mother widening, as one passes from West to East. In this connexion Huxley (1942) says that:

> Genetic polymorphism deserves the most intensive study, especially in cases where the ratios of the types are geographically graded, since here we may hope not only to measure the intensity of the selective forces at work, but to discover also something as to their nature.

Summary

(1) In a composite sample of 1,239 families with 4,139 children, Waterhouse and Hogben found a highly significant shortage of group A offspring in the mating class Father A × Mother O as compared with the reciprocal class Father O × Mother A, and an appreciable shortage of group B offspring in the mating class Father O × Mother B as compared with the reciprocal class Father B × Mother O.

(2) The other mating classes in the Waterhouse-Hogben sample yield some evidence against the possibility that the above loss of group A offspring is caused by the action of maternal anti-A haemagglutinin, the average number of children per family being 3·33 for the 446 heterospecific (*i.e.* serologically incompatible) matings in the sample, and 3·34 in the 793 homospecific matings.

(3) On the other hand, the data give grounds for believing that the shortage of A offspring in the mating class Father A × Mother O is part of a shortage of offspring of A fathers generally, the four types of mating with an A father being less fertile than the four types of mating with a B father. They also give grounds for believing that the shortage of group B offspring in the mating class Father O × Mother B is part of a general shortage of offspring of group B mothers, other than of group B mothers with a group B husband. There are also signs of a loss of group O offspring.

(4) The descending order of fertility of parents in the sample (omitting the symmetric mating classes B × B, O × O, A × A and AB × AB) is B-AB-O-A for fathers, and A-O-AB-B for mothers, the overall descending order being B father, A mother, AB father, O father, O mother, AB mother, A father, B mother. It is suggested that this provides evidence in support of Ford's theory that the ABO groups form a system of balanced polymorphism. Against this theory is the high fertility of B × B and the low fertility of A × A.

(5) It is also suggested that confirmation, modification, or contradiction of the findings would be obtained from a homogeneous population with the same descending order of ABO gene frequency (namely, O-A-B) as that of the diverse Caucasian populations of which the Waterhouse-Hogben data form a sample, or from peoples in whom the descending order is A-O-B or O-B-A.

Addendum

Since this paper went to press it has come to my notice that, in matings with a history of at least two abortions, Sjöstedt and others (1951) found a significant excess of group B wives, other than those with a group B husband. This observation led the authors to remark, "One may have some doubt as to whether the current notion of ABO incompatibility is the right one in the present context".

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References

Allan, T. M. (1952a). *Lancet*, **1**, 102.
—— (1952b). *Ibid.*, **1**, 370.
—— (1953). *Ibid*, **1**, 650.
Bernstein, F. (1924). *Klin. Wschr.*, **3**, 1495.
—— (1925). *S. indukt. Abstamm-.u. VererbLehre*, **37**, 237.
Dungern, E. von, and Hirszfeld, L. (1910). *Z. ImmunForsch.*, **6**, 284.
Fisher, R. A. (1930). "The Genetical Theory of Natural Selection". Clarendon Press, Oxford.
—— (1951). "The Design of Experiments", 6th ed. Oliver and Boyd, Edinburgh.
Ford, E. B. (1948). "Genetics for Medical Students", 3rd ed. Methuen, London.
Glass, B. (1949). *Amer. J. Obstet. Gynec.*, **57**, 323.
Gruhzit, O. M. (1923). *Ibid.*, **5**, 400.
—— (1924). *Ibid.*, **7**, 588.
Hirszfeld, L. (1928). "Konstitutionsserologie und Blutgruppenforschung". Springer, Berlin.
—— (1934). *Ergebn. Hyg. Bakt.*, **15**, 54.
—— (1938). "Les groupes sanguins". Masson, Paris.
—— and Hirszfeld, H. (1919). *Lancet*, **2**, 675.
—— and Zborowski, H. (1925). *Klin. Wschr.*, **4**, 1152.
—— —— (1926). *Ibid.*, **5**, 741.
Hogben, L. (1939). "Nature and Nurture", 2nd ed. Allen and Unwin, London.
Huxley, J. S. (1942). "Evolution. The Modern Synthesis". Allen and Unwin, London.
Koller, S. (1931). *Z. Rassenphysiol.*, **3**, 121.
Landsteiner, K. (1900). *Zbl. Bakt.*, **27**, 357.
—— (1901). *Wien klin. Wschr.*, **14**, 1132.
Levine, P. (1943). *J. Hered.*, **34**, 71.
Mourant, A. E., and Watkin, I. Morgan. (1952). *Heredity*, **6**, 13.
Race, R. R. (1942). *Ann. Eugen., Lond.*, **11**, 365.
Roberts, J. A. Fraser (1951). *Man*, **51**, 94.
Sjöstedt, S., Grubb, R., and Linell, F. (1951). *Acta path. microbiol. scand.*, **28**, 375.
Tovey, G. H. (1945). *J. Path. Bact.*, **57**, 295.
Waterhouse, J. A. H., and Hogben, L. (1947). *British Journal of Social Medicine*, **1**, 1.

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AN INVESTIGATION OF THE RECENT INCREASE IN THE RATE OF NOTIFICATION OF RESPIRATORY TUBERCULOSIS

BY

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In England and Wales during the past decade a paradoxical situation has arisen in that there has been an increase in the number of notifications of respiratory tuberculosis at a time when death rates are falling more rapidly than ever before. The most probable explanation of this phenomenon is the one usually given, namely that the increase in notifications is due to more effective case finding, although the possibility that there has also been an increase in the amount of clinically significant pulmonary tuberculosis cannot be said to have been excluded.

During the 20 years between the two world wars both notification rates and death rates showed a slow but regular improvement. In 1918 more than 30,000 deaths were attributed to respiratory tuberculosis; by 1938 the number had declined by one third to a little over 21,000. In the same period the annual number of notifications fell from 70,000 to less than 38,000, a reduction of almost half. At the beginning of the second world war there was an increase in the annual number of both deaths and notifications, but, whereas the number of deaths soon began to fall again—and at an accelerated rate (only 12,031 persons were reported to have died from respiratory tuberculosis in 1951)—the increase in notifications was more than maintained (42,696 notifications of respiratory tuberculosis were made in 1951, an increase of 5,000 over 1938). The same trend in notification and death rates can be illustrated from recent experience in Scandinavia and America (Stocks, 1952). It is generally believed that the rise in notification and death rates during the early war years reflects an increase in the incidence of tuberculosis brought about by adverse conditions, and that the rapid fall in death rates since the war is to be attributed to advances in therapy.

In the report which follows data collected from records held at the Birmingham Chest Clinic are used in an attempt to discover whether the present high rate of notification is due to a raised incidence of the disease, or to improved case finding.

Secular Trend of Notifications and Deaths

The Figure shows secular variations in the numbers of notifications and deaths from respiratory tuberculosis in Birmingham during the past 30 years. The data have not been expressed as rates because for the war years the related population cannot be estimated reliably. For the present purpose, however, there is little to choose between numbers and crude rates, particularly since between 1931 and 1951 the population of Birmingham has shown only a relatively small increase (from 1,002,603 to 1,112,340) in comparison with the magnitude of the change in number of notifications and deaths. From 1920 until about 1935 notifications decreased rapidly; the number of deaths remained fairly constant until 1933, but fell slightly between 1933 and 1938. During recent years notifications have risen (from 899 in 1940 to 1,242 in 1952). Deaths, on the other hand, after a small increase at the beginning of the war, have continued to fall; indeed, over the past 6 years, the rate of decrease has rapidly accelerated, and the number of deaths reported in 1952 (280) was considerably less than half the number in 1947 (691).

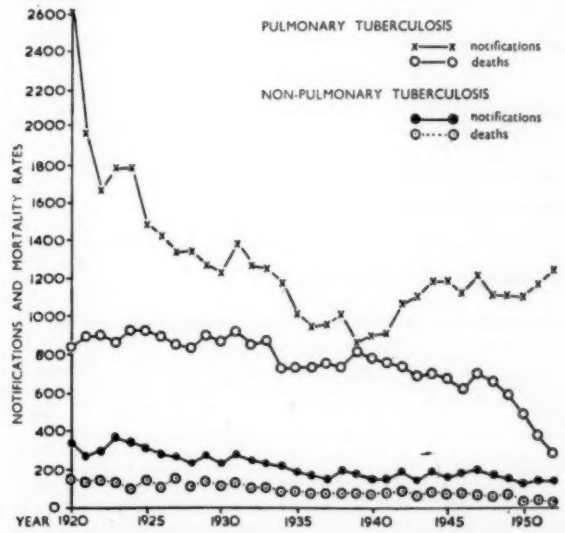

FIGURE.—Notifications of and deaths from tuberculosis in Birmingham, 1920–52

Examination of Reasons for the Increase in Numbers of Notifications

From data held in the records department of the Birmingham Chest Clinic it is possible to relate all new cases of respiratory tuberculosis examined to the source from which they came to the clinic.

Before considering these data, however, we should enquire to what extent new cases examined in any one year at the Chest Clinic can be accepted as representative of all Birmingham notifications during that year (Table I). More than 90 per cent. of all notified cases were examined at least once at the clinic, and in 1952 74 per cent. were notified from the clinic (17 years ago the corresponding proportion was 36 per cent. It is certain, therefore, that any change in the pattern of identification or in the severity of cases of pulmonary tuberculosis notified in Birmingham will be reflected in the new cases examined at the chest clinic.

Between 1940 and 1952 the annual number of new cases seen at the Chest Clinic rose from 877 to 1,132 and we may now consider whether this increase has been associated with any substantial change in case-finding methods. Generally speaking a new patient arrives at an anti-tuberculosis centre by one of four main routes:

(*a*) A statutory notification has already been completed, either by a general practitioner or a hospital physician, and the patient has been asked to attend for confirmatory examination and supervision.

(*b*) The general practitioner has reason to suspect that active tuberculosis may be present, and has sent the patient for specialist opinion.

(*c*) The patient has been in contact with a known case of tuberculosis and has been called to the clinic for routine examination.

(*d*) A suspicious shadow has been detected in the lung fields at mass radiological examination, and the patient has been referred for investigation.

In Table II for representative years between 1935 and 1952 inclusive, all new cases of respiratory tuberculosis examined at the Chest Clinic have been grouped according to the above classification. Between 1940 and 1952 the number of new cases notified before examination at the clinic fell from 496 to 216. This decrease was more than balanced by an increase in both the number and the proportion of new cases derived from each of the other three sources. Although it is evident that mass radiography has played an important part in maintaining high post-war notification rates (for example in 1952 no less than one quarter (280) of all new cases examined had been referred to the Chest Clinic from this service), this is by no means the only

TABLE I

PERCENTAGE OF PERSONS NOTIFIED BECAUSE OF RESPIRATORY TUBERCULOSIS WHO WERE EXAMINED AT CHEST CLINIC

Year			1935	1940	1945	1947	1949	1950	1951	1952
Examined at Chest Clinic	Notified from clinic	%	35·9	42·4	47·8	50·8	60·2	66·0	69·7	73·8
		No.	367	381	570	621	682	748	825	916
	Notified before reaching clinic	%	58·0	55·2	46·6	34·8	31·7	31·4	21·5	17·4
		No.	593	496	556	426	359	356	254	216
Not examined at Chest Clinic		%	6·2	2·4	5·6	14·4	8·1	2·6	8·9	8·9
		No.	63	22	67	176	92	29	105	110
All Notifications in Birmingham		%	100	100	100	100	100	100	100	100
		No.	1,023	899	1,193	1,223	1,133	1,133	1,184	1,242

TABLE II

SOURCE OF NEW CASES OF RESPIRATORY TUBERCULOSIS SEEN AT CHEST CLINIC

Year		1935	1940	1945	1947	1949	1950	1951	1952
Notified	%	61·8	56·6	49·4	40·8	34·5	32·2	23·5	19·1
	No.	593	496	556	426	359	356	254	216
Referred as Suspects	%	29·8	36·9	43·5	47·4	54·6	47·6	47·5	46·6
	No.	286	324	490	496	568	525	512	527
Examined as Contacts	%	8·4	6·5	3·6	8·4	7·5	10·6	10·8	9·6
	No.	81	57	41	88	78	117	117	109
Referred from Mass *X* Ray Service	%	—	—	3·5	3·5	3·5	9·6	18·2	24·7
	No.	0	0	39	37	36	106	196	280
TOTAL	%	100	100	100	100	100	100	100	100
	No.	960	877	1,126	1,047	1,041	1,104	1,079	1,132

reason for the rise in notifications. The number of new cases derived from examination of contacts also increased (from 57 in 1940 to 109 in 1952), and the number of suspects found to have respiratory tuberculosis rose from 324 to 527.

The increase in the number of suspects and contacts found to have pulmonary lesions may at first sight suggest that there has been a rise in incidence of respiratory tuberculosis among the general population from which they were drawn. Table III to some extent modifies this impression. Between 1940 and 1952 the annual number of suspects examined at the Clinic increased from 1,928 to 5,618, and the number of contacts from 1,041 to 5,577; the percentages found to have pulmonary lesions fell from 16·8 to 9·4 and from 5·5 to 2·0 for the two groups respectively. That is to say that, while the number of examinations of individuals from these sources increased more than four times, the number of cases of pulmonary tuberculosis identified was not quite doubled. In passing, it is of some interest to note that although by 1952 the number of patients notified before examination at the Chest Clinic had fallen to less than half the number of 17 years ago, more than 30 per cent. of notifications were found to be incorrect.

The decline in the proportion of notifiable lesions among patients sent to the Chest Clinic as suspects does not by itself help us to arrive at any conclusion concerning changes in the incidence of tuberculosis in the general population, since it may be no more than an expression of an increased awareness of the value of early diagnosis and treatment by patients and their general practitioners. The fall in incidence among contacts examined at the Clinic, however, is more suggestive of a general fall in the incidence of clinically significant tuberculosis.

There is, of course, a possible alternative explanation. The remarkable increase between 1940 and 1952 in the annual number of contacts examined indicates that in the earlier years contact tracing was far from complete, and in these circumstances it is probable that the service was to some extent focused on families where the risk might be expected to have been greatest. But we can probably dismiss this possibility on the evidence presented in Table IV,

TABLE III

PERCENTAGE OF ALL NEW PATIENTS EXAMINED AT CHEST CLINIC WHO WERE FOUND TO HAVE RESPIRATORY TUBERCULOSIS

Year		1935	1940	1945	1947	1949	1950	1951	1952
Notified	%	67·3	71·9	70·3	66·9	66·1	69·9	63·8	69·5
	No.	881	690	791	637	543	509	398	311
Referred as Suspects	%	18·3	16·8	11·3	9·8	8·4	8·7	8·6	9·4
	No.	1,559	1,928	4,354	5,064	6,796	6,027	5,964	5,618
Examined as Contacts	%	7·2	5·5	4·2	3·9	3·0	3·3	3·8	2·0
	No.	1,131	1,041	966	2,255	2,585	3,523	3,085	5,577
Referred from Mass *X* Ray Service	%	—	—	22·9	26·2	48·6	54·9	58·5	53·1
	No.	—	—	170	141	74	193	335	527
TOTAL	%	26·9	24·0	17·9	12·9	10·5	10·8	11·0	9·4
	No.	3,571	3,659	6,281	8,097	9,928	10,252	9,782	12,035

TABLE IV

PERCENTAGE GROUP I AMONG NEW CASES OF RESPIRATORY TUBERCULOSIS EXAMINED AT CHEST CLINIC RELATED TO SOURCE

Year		1935	1940	1945	1947	1949	1950	1951	1952
Notified		12·5	14·3	30·4	30·8	23·7	28·6	31·1	41·2
Referred as Suspects		27·6	20·1	24·1	24·4	23·4	21·5	30·3	33·4
Examined as Contacts		59·3	57·9	70·7	52·3	53·8	47·9	53·0	56·1
Referred from Mass *X* Ray Service		—	—	48·7	32·4	30·1	25·5	37·2	28·8
TOTAL	%	20·9	19·3	29·8	29·6	26·0	26·8	34·0	35·8
	No.	960	877	1,126	1,047	1,041	1,104	1,079	1,132

TABLE V
CLASSIFICATION OF NEW CASES OF RESPIRATORY TUBERCULOSIS SEEN AT CHEST CLINIC

Year		1935	1940	1945	1947	1949	1950	1951	1952
Group I	%	20·9	19·3	29·8	29·6	26·0	26·8	34·0	35·8
	No.	201	169	335	310	271	296	367	406
Group II	%	43·0	38·5	41·0	44·4	51·5	50·4	46·6	48·1
	No.	413	338	462	465	536	556	503	544
Group III	%	36·0	42·2	29·2	26·0	22·5	22·8	19·4	16·1
	No.	346	370	329	272	234	252	209	182
TOTAL	%	100	100	100	100	100	100	100	100
	No.	960	877	1,126	1,047	1,041	1,104	1,079	1,132

which shows the proportion of early cases (Group I) among new cases of respiratory tuberculosis identified at the clinic. Between 1940 and 1952, among all new cases, the percentage in Group I increased from 19·3 to 35·8; but among contacts there was no appreciable change.

DISCUSSION

The data examined above provide support for the view that the recent rise in notification rates is due to more successful case finding, rather than to a raised incidence of the disease. The increase in the number of new cases discovered between 1940 and 1952 was due to mass radiography, which came into use during these years, and to an increase (by about four times) in the number of persons examined at the Chest Clinic as suspects and contacts. In the same period the proportion of suspects and contacts found to have clinically significant tuberculosis fell very considerably. While the evidence in the case of suspects is hard to interpret, the reduced incidence among contacts strongly suggests that the incidence of the disease must also have fallen and certainly indicates that it has not risen. It is hardly correct to attribute the more successful case finding wholly to mass radiography (as was done in the Annual Report of the Ministry of Health, 1951), since about half the increase between 1940 and 1952 was due to the extension of the work among suspects and contacts.

Finally, it may be noted that the increase in the number of cases discovered is associated with an increase in the proportion of early cases (Table V). During the 13 years considered the number of early (Group I) cases examined at the clinic increased nearly three times (from 169 to 406), while the number of late (Group III) cases fell by half (from 346 to 192). Since early respiratory tuberculosis is more amenable to treatment, it seems certain that more intensive methods of case finding have made a considerable contribution to the recent decline in mortality rates, which cannot be attributed wholly to advances in therapy.

SUMMARY

(1) During recent years in Birmingham, as in England and Wales, the number of notifications of respiratory tuberculosis has increased while the number of deaths has fallen. Data collected from the Birmingham Chest Clinic (where more than 90 per cent. of all Birmingham notifications are examined) are used to inquire whether the high rate of notification is due to a raised incidence of the disease or to improved case finding.

(2) It is shown that between 1940 and 1952 the number of new cases notified before examination at the Clinic fell from 496 to 216. The increase in the number of notifications from all sources (from 877 to 1,132) is due about equally to:

(*a*) cases discovered by mass radiography (280 in 1952),

(*b*) an increase in cases identified among suspects and contacts (from 381 to 636).

(3) In the same period, the proportion of patients with notifiable lesions discovered among suspects and contacts examined at the Clinic fell from 17 per cent. to 9 per cent. and from 6 per cent. to 2 per cent. respectively. The fact that the number of new cases from these two sources was almost doubled is explained by the fact that four times as many persons were examined.

(4) These observations strongly support the accepted view that the raised incidence of notification is due to more effective case finding.

We acknowledge with pleasure the facilities afforded to us by Dr. Matthew Burn, Medical Officer of the City of Birmingham, and the assistance of Mr. L. Barratt in finding our way through the Birmingham Chest Clinic records.

REFERENCES

Ministry of Health (1953). "Report for the Year 1951", Part III. H.M.S.O., London.

Stocks, P. (1952). *In* "Modern Practice in Tuberculosis", ed. T. H Sellers and J. L. Livingstone, vol. 1, pp. 8–27. Butterworth, London.

Brit. J. prev. soc. Med. (1953), **7**, 231–239

PHYSIQUE AND DEVELOPMENT OF BOYS AND GIRLS FROM DIFFERENT TYPES OF SCHOOL

BY

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From the Ministry of Health, London

This paper is a report on certain aspects of a wide investigation into the physique and growth of schoolchildren undertaken from 1945 to 1952, with special reference to age, sex, and social differentia. The particular purpose of this part of the study was to compare the physique of groups of boys and girls from three different types of school representing different socio-economic levels; Group A consisting of children from independent (public or preparatory) schools, Group B children from council schools in the best neighbourhoods of a number of industrial towns, and Group C children from council schools in the poorest (slum) areas of these towns.

MATERIAL.—It was impracticable to obtain children of these groups by random selection. Schools were therefore selected, in consultation with the Medical Officer of Health of the district, as most likely to be representative of the three groups. In most cases all the pupils of the required ages were measured, *i.e.* there was no selection within the schools. Ages range from 5 to 18 years for Group A, 5 to 15 for Group C, and from 7 to 14 for Group B. For the present purpose the age group 10 years includes all children who have attained the age of 10 but who have not yet reached the age of 11.

Apart from a trial group to determine the most appropriate measurement, some 1,430 independent school children and 1,495 public elementary school children were measured, giving approximately fifty children of each age, sex, and type of school. A team of three* measured all the children, each member being responsible for particular measurements throughout.

Three accuracy trials were carried out, involving full repeat measurement of 190 children. The results can be summarized very briefly by giving the error variances as a percentage of the total variance for each measurement at each age and sex. The order of accuracy on this basis may be tabulated as in previous column.

Mean measurements for Groups A and C (the most complete) at each age are shown for boys and girls in Appendix Tables A and B. Full details of the various techniques, together with standard deviations of the measurements and the means for Group B, may be obtained from the author.

Measure	Site	Percentage
Weight	Total	0·2
Girth	Arm	0·7
	Knee, ankle	1
	Wrist	3
	Chest, hip	4
	Waist	6
Length	Total height, arm	1
	Leg	2
	Sitting height	3
	Foot	5
	Hand	6
Breadth	Inter-trochanteric	3
	Hand	7
	Inter-spinous, inter-acromial ..	10
	Foot	12

I. INCREASE IN SIZE WITH AGE

(*a*) *Increase in Absolute Measurements.*—The measurements can be divided into two groups according to their association with age.

Type I measurements (height, arm and leg length, foot and hand length and breadth, and inter-acromial, inter-spinous, and ankle breadth) show a steady increase with age (Fig. 1*a*).

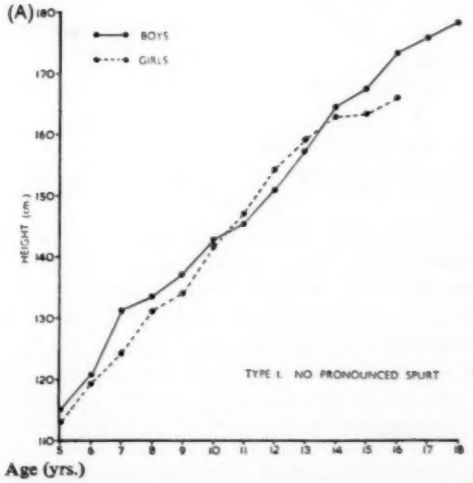

FIG. 1(*a*).—Measurements according to age. Type I Height (no pronounced spurt) by sex. Independent schools (Group A).

* Dr. E. W. Adcock, Miss J. Stevenson, and the author.

Type II measurements (weight, trunk, knee, arm, and wrist girth, sitting height, and inter-trochanteric breadth) show a pattern of slow increase through early childhood followed by a spurt just before and during puberty with gradual reduction in rate from age 15 onwards (Fig. 1*b* and *c*). The effect of sex on these two types of measurement is also noted below.

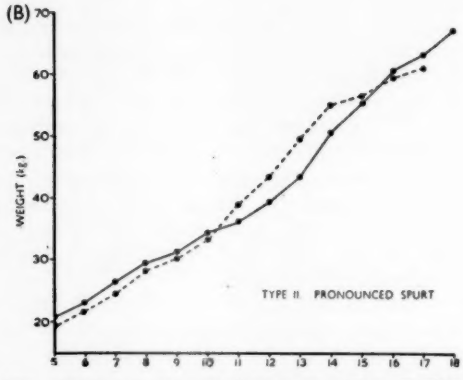

FIG. 1(*b*).—Measurements according to age. Type II Weight (pronounced spurt) by sex. (Independent Schools).

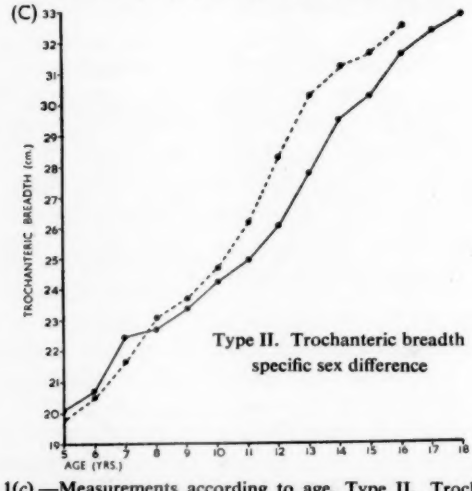

Fig. 1(*c*).—Measurements according to age. Type II. Trochanteric breadth. Specific sex difference. Independent schools.

The differences between successive annual means in weight, height, major lengths, and acromial breadth are significant at all ages. Foot and hand measurements give significant differences at nearly all ages, but trunk and limb girths are more variable, and differences are least significant from 9–10 years in girls and 10–11 years in boys, *i.e.* just before the growth spurts.

(*b*) *Inter-relationships between Measurements.*—Apart from the differences in absolute measurements which occur with age, we find different relative rates of development which can be shown by plotting other measurements against height or weight (Figs 2 and 3). When weights or girths are plotted vertically and lengths horizontally, a concave curve results, showing that for equal changes in length, girths and weights increase slowly at first and then at a gradually accelerated rate, in some cases until age 17 or 18, in others until age 15. When other lengths are compared with height they give straight line or a slightly concave curve, *i.e.* the increase is proportional to height, or is greater in relation to height in short (*i.e.* in young) than in older children. Similarly, when compared with weight, girths show an almost

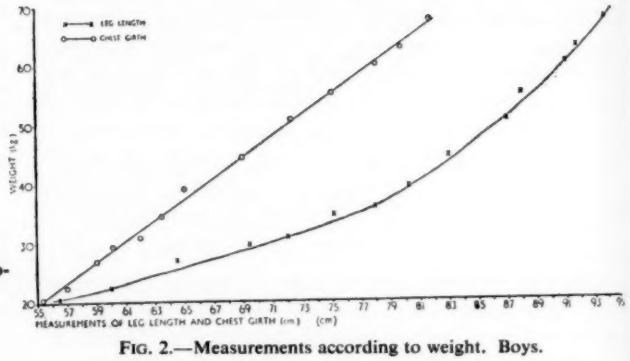

FIG. 2.—Measurements according to weight. Boys.

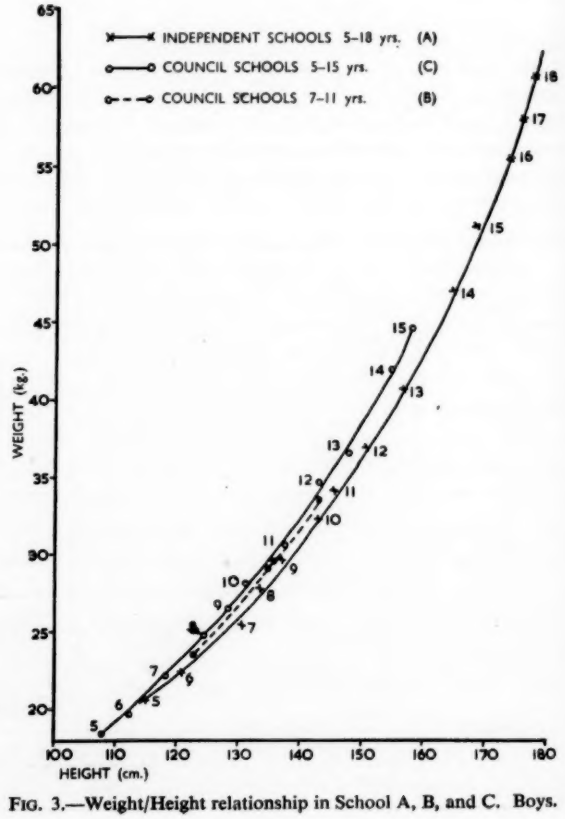

FIG. 3.—Weight/Height relationship in School A, B, and C. Boys.

straight line, *i.e.* girths and weights increase in proportion with each other. Differences from the straight line tend to be greater for boys than for girls as boys' rates vary more with age. If, however, heights or lengths are plotted against the logarithm of weight, they also show an almost straight line relation (Figs 4*a* and *b*).

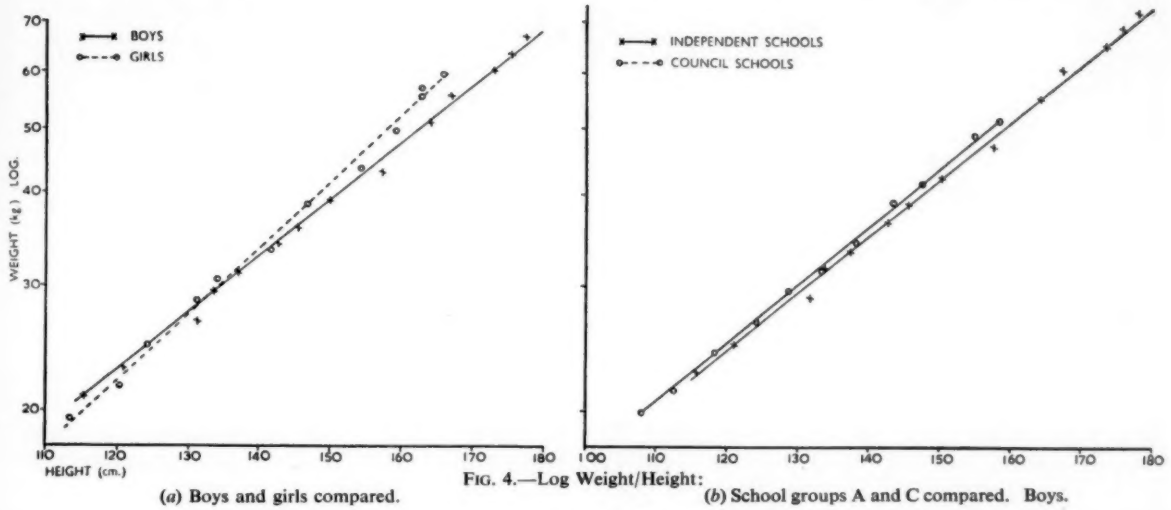

FIG. 4.—Log Weight/Height:
(*a*) Boys and girls compared. (*b*) School groups A and C compared. Boys.

If the lines are parallel for two groups they indicate constant differences in proportions (stockiness or slenderness) and if they diverge they indicate that one group is increasing in girth faster than the other. To determine the significance of individual or group differences requires the calculation of the variability of individual log-weight/height values for a representative child population rather than for two selected groups such as the present ones.

(*c*) *Time of Maximum Growth.*—Other differences in development of the various measurements are found by considering the proportions of the final or mature stage reached by each type of measurement at different ages. On the average, apart from weight, the measurements of boys at age 18 are 1·5 times those at age 5 (1·4 to 1·6). Similarly the measurements of girls at 16 years average 1·4 times those at age 5 (1·3 to 1·6). If for any measurement the growth from age 5 to any intervening age is expressed as a percentage of the total growth from 5 to 18 years, the measurements form two groups consisting of hand length, breadth, height, leg length, and acromial breadth on the one hand, and trochanteric girth, knee girth, arm girth, and trunk girth, on the other (Fig. 5, overleaf), the latter lagging behind until age 10 and then catching up.

It may be fruitful to compare the progress of *groups* of individuals in the same measurements in respect of the distance travelled along the road to maturity; especially in cases where direct comparison of size may be complicated by genetic differences between the different social classes. By extending the curves backwards to the probable birth weights and lengths (accepting mean values for boys and girls of 7·5 and 7·3 lb. and 50 and 49 cm. for both social groups, social differences at birth being comparatively negligible) and forwards to the adult state*, and using similar data which we have obtained for younger ages, we can interpret the present data according to the scale of total growth from birth to maturity (Fig. 6, overleaf). Differences are compared in detail under the sections concerned with sex and social differences. This procedure can, of course, be used only to interpret results observed after maturity, unless data for the adult conditions are obtained from some other comparable group and used as the standard.

II. Sex Differentials in Size and Growth

(*a*) *Increase in Absolute Measurements.*—Appendix Tables A and B show that only four measurements (arm length, foot length and breadth, and hand breadth) are greater for boys than for girls at all ages. It is significant that these measurements are those in which there is no pronounced pre-pubertal spurt (Fig. 1*a*). In all other measurements, the pattern is that boys' measurements slightly exceed girls' measurements until age 10, when the girls begin to grow at a faster rate and overtake the boys.

* At age 18 to 19 growth in linear measurements has almost ceased. Kemsley (1952) shows that the mean height increases less than 1 per cent. after 18 years, so that present results for Group A can be accepted as a close approximation to the adult level and Group C's adult height can be taken as somewhat lower than that of the general population. Similarly, the probable weights for the two groups when adult can be estimated.

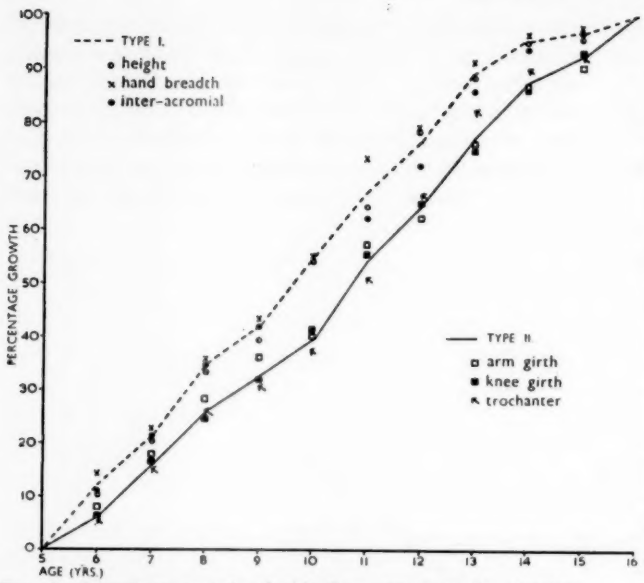

FIG. 5.—Percentage growth of girls from age 5 to 16, showing Type I height, etc. and Type II girths.

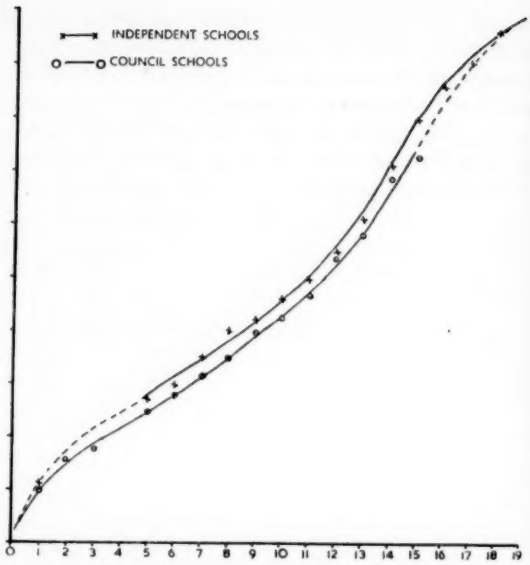

FIG. 6.—Percentage growth of boys from birth to age 18, by weight, comparing Schools A and C.

They remain ahead until age 14 by which time the later, and usually more intensive, pre-pubertal spurt of the boys enables them to pass the girls. Boys continue to grow for a longer period than girls, and hence the gap widens until the adult stage is reached (Fig. 1*b*). In only two measurements is this pattern modified; in pelvic breadth (whether of spinous breadth or trochanteric breadth) and pelvic girth* girls' measurements continue to exceed those of boys up to age 17. This represents a specific sex difference as distinct from one of all round growth (Fig. 1*c*).

(*b*) *Inter-relationship between Measurements.*—The slopes for boys and girls show, for example, that hip girth increases more for corresponding weight increases in girls than in boys, and that the converse obtains for leg length. Similarly, differences in the *level* of the curves show for which sex the different measurements are more prominent (*i.e.* which measurements contribute the bigger proportion to weight or height).

The straight lines for log weight and height cross in the diagram, the slopes being steeper for boys than for girls, showing that boys become taller relative to weight after 130 cm. height (at age 8 or 9) and that they become progressively less stocky compared with girls.

It has already been noted that by age 10 girls have accomplished a greater proportion of their total growth in length and girth than boys; this difference is greater by age 11 or 12 when boys have not yet made their major spurt in growth (especially as regards girth).

* The comparison of girth applies to Group B and the follow-up measurements, but not to the measurements shown in the Appendices since the maximum (seat) girth was not taken initially for boys.

III. Differences between Individuals from Three Types of School

(*a*) *Increase in Absolute Measurements.*—If we compare the mean measurements for Groups A and C at each age, we find the number of annual groups in which measurement differences are significantly in favour of the independent schools ($2 \times 5{\cdot}2$) are as shown in Table I. There is considerable size difference in favour of the independent school groups

TABLE I

NUMBER OF COMPARISONS IN WHICH INDEPENDENT SCHOOL MEANS EXCEED COUNCIL SCHOOLS

Measurements	Number of Comparisons	
	Girls (16)	Boys (17)
Weight (kg.)	14	17
Standing height	16	17
Sitting height	16	16
Leg length	16	17
Arm length	14	17
Foot length	16	16
Foot breadth	7	5
Hand length	9	9
Hand breadth	9	9
Chest girth	6	12
Waist girth	5	6
Hip girth	11	11
Arm girth	11	11
Wrist girth	12	7
Ankle girth	14	17
Knee girth	15	14
Inter-spinous	13	15
Inter-trochanteric	14	13
Bi-acromial	15	14

at all ages, especially in length measurements. The superiority varies from 1 to 3 years' growth, but differs from one measurement to another, being least marked in girths, especially in waist girth among girls, where Group A results are actually somewhat lower than Group C results below age 11.

(*b*) *Inter-relationship between Measurements*—We can again plot mean heights and weights on a graph this time comparing the social groups (Fig. 3). The resulting values form parabolic curves, from which much useful information can be obtained. For example, the fact that different weight/height curves are given for the social groups shows that the difference is not merely one of overall size. The horizontal distance between the curves at any point indicates the amount by which the independent school children are taller than the council school children for any given weight, whilst the vertical distance indicates the weight superiority of the council school group compared with independent school children of the same height. The difference between the age points along the two curves amounts to about 2 years' growth at ages 7 to 10.

The particular merit of the log weight/length relation is that, being a straight line, the curve is independent of the size of the variables and therefore of age.

IV. Physical Maturity or Developmental Stage

In the same way as sex or social differences are represented by the *pattern* of present measurements as well as by their size, so physical maturity is shown by a changing pattern of measurements with age, *i.e.* by a change in body form, in addition to increase in size. If we express each measurement as a proportion of the total of the measurements (excluding weight) for the particular age, we can indicate the change of form very simply by the sum of those measurements which increase their proportion with age compared with those that decrease. The resultant values (Table II) change sufficiently regularly to enable a straight line to be fitted. This means that the level of physical maturity or developmental age can be indicated for a group or for an individual by reading off the age corresponding to the appropriate shape value on the straight line, in much the same way as Todd's standards of skeletal maturation are used by matching individuals against the standards. Separate curves have been produced for the sexes and school groups.

The results show that the independent School children are more advanced for their age than the council school children by amounts up to 3 years in respect of their "shape age". This confirms other indications of the maturity differences of the groups. However, the actual values depend upon the particular combination of measurements used in the present study. A simpler and more generally applicable device is desirable and may be applied as follows.

TABLE II

CHANGE OF SHAPE ACCORDING TO AGE AND SOCIAL GROUP

Sex	Age (yrs)	Schools	
		Council	Independent
Boys	5	·2945	·3016
	6	·2993	·3066
	7	·3023	·3083
	8	·3054	·3123
	9	·3072	·3146
	10	·3090	·3165
	11	·3120	·3198
	12	·3133	·3207
	13	·3154	
Girls	5	·4000	·4072
	6	·4005	·4096
	7	·4053	·4114
	8	·4080	·4135
	9	·4097	·4166
	10	·4112	·4195
	11	·4137	·4201
	12	·4169	·4281
	13	·4191	

Boys = Sum of contribution of height, leg, and inter-trochanteric breadth as proportion of total of measurements (items 2–19 in Appendix I).

Girls = Sum of contributions of height, leg length, hip, and knee.

In infancy trunk length contributes the main proportion of total length, whereas at later ages the legs contribute an increasing proportion. Therefore the ratio of sitting height to leg length (Fig. 7) can also be used to indicate maturity (especially since the two coefficients of variation are comparable at all ages). These values are set out for both boys and

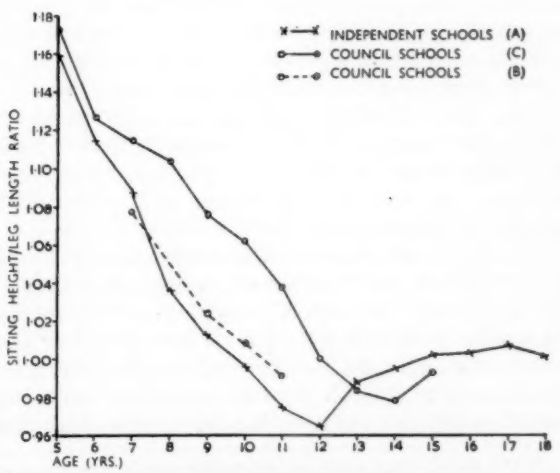

FIG. 7.—Ratio of sitting height to leg length of boys from age 5 to 18, comparing Schools A, B, and C.

girls in Table III.* Variability of the values is high and one should not expect individuals to follow this pattern, though groups tend to do so.

TABLE III
RATIO OF SITTING HEIGHT TO LEG LENGTH ACCORDING TO SEX AND AGE

Age (yrs)	Boys		Girls	
	Independent Schools	Council Schools	Independent Schools	Council Schools
4	—	1·219	—	—
5	1·155	1·174	1·133	1·148
6	1·116	1·126	1·095	1·123
7	1·100	1·118	1·078	1·099
8	1·036	1·107	1·038	1·078
9	1·013	1·078	1·021	1·058
10	0·999	1·061	1·000	1·055
11	0·973	1·036	0·990	1·031
12	0·962	1·000	0·991	1·016
13	0·987	0·982	1·005	1·010
14	0·993	0·979	1·018	1·014
15	1·001	0·991	1·020	—
16	1·000	—	1·017	—
17	1·006	—	1·007	—
18	0·998	—	—	—

V. Effect of Differential Development Rates on Group Measurements

Differences in size between children in different social groups increase with age, but important differences are already present at age 5, when boys in Group A have the weight of an average 6-year-old child, while those in Group C have that of the average for 4 to 5 years. If we accept the differences between the social groups as due to differences in developmental age, we should expect, at the next year's measuring, that Group A would have increased by the average growth increment for age 6–7 compared with C's increment from 4 to 5. If the same holds good at later ages the increment would always be 1–2 years in advance. Since the annual increments increase in size from year to year this would involve a cumulatively greater difference between the groups in actual size as age increases. If we set out the weights which would result in Group A, assuming the annual progression to be one year ahead of average and beginning with the attained weight of 46 lb. (20·9 kg.) at age 5, and similarly those which would be found in Group C one year behind the average, we find the agreement with the actual weights set out in Table IV.

With girls the difference in measurements is not apparent until age 8, when the independent school group exceeds the council school group by about 1½ years. If, therefore, we treat the results in the same way, allowing the increments appropriate to the two ages, we find the correspondence with the actual weights set out in Table IV. Had we assumed this difference to have begun at age 5 we should have expected a higher weight for the independent school group at that age than the weight actually found, but the results would have been the same from age 8 onwards. Thus the agreement in girls is not quite so close as in boys.

TABLE IV
COMPARISON OF ACTUAL AND EXPECTED WEIGHTS OF BOYS AND GIRLS

Sex	Age (yrs)	Weight (kg.)			
		Independent Schools		Council Schools	
		Expected	Actual	Expected	Actual
Boys	5	20·9	20·9	18·4	18·4
	6	24·0	22·8	19·7	19·9
	7	26·5	26·5	21·4	22·4
	8	28·7	29·3	24·5	24·7
	9	31·2	31·1	26·9	27·3
	10	33·5	34·0	29·2	29·4
	11	37·4	36·1	31·7	31·9
	12	40·6	39·3	34·0	36·5
	13	47·5	43·5	37·9	38·8
	14	51·0	50·8	41·1	45·3
	15	55·8	55·5	47·9	47·6
Girls	8	28·4	28·4	23·8	23·8
	9	31·2	30·4	26·2	26·4
	10	35·4	33·6	28·5	28·8
	11	39·7	39·0	31·3	31·7
	12	45·8	43·8	35·5	35·6
	13	49·7	49·8	39·8	41·8
	14	53·6	55·1	45·9	44·2

VI. Discussion

When considering some twenty body measurements in two sexes, and three social groups, by annual age groups, the results can be somewhat confusing unless they can be reduced to patterns. In the first place differences between absolute measurements are of little interest, although differences in their change with sex, age, or social group may be. In respect of change with age the measurements can be divided into two types:

Type I. *Most Lengths and Some Breadths.*—There is an almost straight-line increase in size from age 5 to 14 or 15 (boys) and from 5 to 13 (girls), followed by a slowing down in the rate of growth

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From age 5 to 18 the fact that the logarithm of weight is proportional to height shows that weight is acting as a cubic function of a linear measurement (actually to the power 3·14); this applies also to major girths. Weight and height (which represent the two measurement groups) have been extended to include results for birth and maturity, and over this period three phases of post-natal growth can be distinguished. They may be summarized in the phraseology of Tanner (1951) as:

Phase 1 (birth to 5 years) high velocity but decreasing acceleration;

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Boys show this relative pause in growth and subsequent surge much more strongly than girls. In fact, girls show no "pubic spurt" in most of their measurements. The characteristics of these two groups of measurements affect and possibly account for some of the differences between the sexes and social groups, *i.e.* girls' measurements are generally lower than those of boys up to age 11, and they then exceed those of boys until the boys overtake them again at age 15. This is mainly because the girls' Phase 2 is telescoped into a shorter period. Moreover, the differences are smaller in those measurements (Type I) which have an almost constant increment, and greatest in those in which growth increases most rapidly with age (Type II).

It is desirable to have a practical indication of maturity from body measurements apart from the estimation of skeletal maturity by *x* rays (which is inconvenient to carry out, and in any case only applies to one system); by estimating the position of a group along its development curves in particular measurements, it is possible to gain suggestive evidence of the stage of maturity of the group. The maturity—in the sense of distance along these curves—may, of course, be different in different measurements, and one aim of this paper has been to show how, not the absolute size, but the relation between measurements (*i.e.* shape) or position along the age curve (*i.e.* relation between present position and the previous and subsequent positions) can be used for this purpose.

The school groups show differences in general size, but the independent school group has also the more slender build which characterizes greater maturity, the shape (based either on all measurements or on the proportion of trunk to limb ratio) of more mature ages, and the pattern of weight increase appropriate to a group 2 years older. All these factors point to an increase in the rate of maturation of the independent school group, possibly superimposed on general size differences and an overall difference in body build.* It is possible that the different mature physiques of the social groups are due to these differences in the rate of development rather than to specifically social differences. This would imply that children of the lower social group whose development was ahead of the average would tend to converge upon the characteristics of the higher social group.

VII. Summary

The present paper reports the results of analysing cross-sectional body measurements of boys and girls aged 5 to 18 years from three different types of school representing different socio-economic classes.

Body measurements of boys and girls between 5 and 18 years can be classified into two types according to the way in which they increase throughout the period. Type I (consisting mainly of lengths) show an almost constant increase during the period 5 to 15 years (boys) and 5 to 13 years (girls), and Type II (girths and some breadths) show slower growth during the period 5 to 10 years followed by the more rapid growth of the "pubic spurt". Similarly, there is a lag in the development of Type II measurements, whereby the corresponding proportion of the mature size is reached one year later than for Type I measurements because of the relative pause in growth of Type II measurements before the pubertal increase.

When weight or girths are set against lengths over the whole period a smooth curve results, and this can be transformed to a straight line by using a log scale for the girths.

The proportions of the total measurement contributed by each measurement tend to vary in a regular fashion with age, and these proportions have been expressed in the form of a straight line equation.

A similar function which varies regularly with age is the relation between sitting height and leg length. Both these functions can be used to indicate a "shape age".

The sexes can be compared in respect of any of the above. Thus girls show little evidence of the pubic spurt which occurs in Type I measurements for boys. The slopes of the log weight/height lines

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girls in Table III.* Variability of the values is high and one should not expect individuals to follow this pattern, though groups tend to do so.

TABLE III
RATIO OF SITTING HEIGHT TO LEG LENGTH ACCORDING TO SEX AND AGE

Age (yrs)	Boys		Girls	
	Independent Schools	Council Schools	Independent Schools	Council Schools
4	—	1·219	—	—
5	1·155	1·174	1·133	1·148
6	1·116	1·126	1·095	1·123
7	1·100	1·118	1·078	1·099
8	1·036	1·107	1·038	1·078
9	1·013	1·078	1·021	1·058
10	0·999	1·061	1·000	1·055
11	0·973	1·036	0·990	1·031
12	0·962	1·000	0·991	1·016
13	0·987	0·982	1·005	1·010
14	0·993	0·979	1·018	1·014
15	1·001	0·991	1·020	—
16	1·000	—	1·017	—
17	1·006	—	1·007	—
18	0·998	—	—	—

V. Effect of Differential Development Rates on Group Measurements

Differences in size between children in different social groups increase with age, but important differences are already present at age 5, when boys in Group A have the weight of an average 6-year-old child, while those in Group C have that of the average for 4 to 5 years. If we accept the differences between the social groups as due to differences in developmental age, we should expect, at the next year's measuring, that Group A would have increased by the average growth increment for age 6–7 compared with C's increment from 4 to 5. If the same holds good at later ages the increment would always be 1–2 years in advance. Since the annual increments increase in size from year to year this would involve a cumulatively greater difference between the groups in actual size as age increases. If we set out the weights which would result in Group A, assuming the annual progression to be one year ahead of average and beginning with the attained weight of 46 lb. (20·9 kg.) at age 5, and similarly those which would be found in Group C one year behind the average, we find the agreement with the actual weights set out in Table IV.

With girls the difference in measurements is not apparent until age 8, when the independent school group exceeds the council school group by about 1½ years. If, therefore, we treat the results in the same way, allowing the increments appropriate to the two ages, we find the correspondence with the actual weights set out in Table IV. Had we assumed this difference to have begun at age 5 we should have expected a higher weight for the independent school group at that age than the weight actually found, but the results would have been the same from age 8 onwards. Thus the agreement in girls is not quite so close as in boys.

TABLE IV
COMPARISON OF ACTUAL AND EXPECTED WEIGHTS OF BOYS AND GIRLS

Sex	Age (yrs)	Weight (kg.)			
		Independent Schools		Council Schools	
		Expected	Actual	Expected	Actual
Boys	5	20·9	20·9	18·4	18·4
	6	24·0	22·8	19·7	19·9
	7	26·5	26·5	21·4	22·4
	8	28·7	29·3	24·5	24·7
	9	31·2	31·1	26·9	27·3
	10	33·5	34·0	29·2	29·4
	11	37·4	36·1	31·7	31·9
	12	40·6	39·3	34·0	36·5
	13	47·5	43·5	37·9	38·8
	14	51·0	50·8	41·1	45·3
	15	55·8	55·5	47·9	47·6
Girls	8	28·4	28·4	23·8	23·8
	9	31·2	30·4	26·2	26·4
	10	35·4	33·6	28·5	28·8
	11	39·7	39·0	31·3	31·7
	12	45·8	43·8	35·5	35·6
	13	49·7	49·8	39·8	41·8
	14	53·6	55·1	45·9	44·2

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When weight or girths are set against lengths over the whole period a smooth curve results, and this can be transformed to a straight line by using a log scale for the girths.

The proportions of the total measurement contributed by each measurement tend to vary in a regular fashion with age, and these proportions have been expressed in the form of a straight line equation.

A similar function which varies regularly with age is the relation between sitting height and leg length. Both these functions can be used to indicate a "shape age".

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cross for boys and girls, as the boys become more slender than girls with age.

The independent school children (Group A), besides being on the average 2 years ahead of the council school children (Groups B and C) in size, are also more slender and have the shape characteristics of at least 2 years ahead. The increases in weight of Group A between successive years' measurements also follow a pattern which is approximately 2 years in advance of Group C. Thus, the evidence is strongly in favour of a difference in maturation rate as a prime cause of the social differences noted.

I wish to acknowledge the help of Dr. E. W. Adcock and Miss J. Stevenson in the field work, of the Medical Officers of Health and School Medical Officers of the various districts for their ready co-operation, of the Heads and Staffs of the schools for their hospitality and tolerance, and of Dr. E. R. Bransby, whose helpful discussion at every stage of the work was more in the nature of collaboration than help.

REFERENCES

Berry, W. T. C., and Cowin, P. J. (1951). *Med. Off.*, **85**, 213.
Daley, A. (1950). "Report on the Heights and Weights of School Pupils in the County of London in 1949." London County Council.
Kemsley, W. F. F. (1952). *Ann. Eugen., Camb.*, **16**, 316.
Leitch, I. (1951). *Brit. J. Nutr.*, **5**, 142.
Tanner, J. M. (1951). *Hum. Biol.*, **23**, 93.

APPENDIX TABLE A

MEAN MEASUREMENTS (cm.) BOYS (see Notes on next page)

Age (yrs)		5	6	7	8	9	10	11	12	13	14	15	16	17	18
Weight (kg.)	A	20·9	22·8	26·5	29·3	31·1	34·0	36·1	39·3	43·5	50·8	55·5	60·3	63·1	67·0
	C	18·4	19·9	22·4	24·7	27·3	29·4	31·9	36·5	38·8	45·3	47·6	—	—	—
Standing Height	A	115·2	121·0	131·4	133·6	137·3	142·9	145·4	151·1	157·3	164·4	167·2	173·3	175·9	178·3
	C	108·0	112·6	118·4	124·1	128·8	133·7	138·1	143·5	147·8	155·0	158·5	—	—	—
Sitting Height	A	65·6	67·5	70·3	70·3	72·6	74·1	74·9	77·5	81·9	86·6	88·2	91·1	92·6	93·5
	C	61·4	63·4	66·4	69·2	70·7	72·6	74·1	75·8	77·3	80·5	83·4	—	—	—
Leg Length	A	56·8	60·5	64·6	69·9	71·4	75·1	77·9	80·6	83·0	87·2	88·1	91·1	92·0	93·6
	C	52·3	55·3	59·4	62·5	65·6	68·4	71·5	75·8	78·7	82·2	84·1	—	—	—
Arm Length	A	46·3	49·0	52·8	56·6	58·2	60·6	61·4	64·4	66·0	69·0	70·7	73·5	73·2	76·0
	C	45·1	47·1	49·7	51·9	54·2	55·9	57·9	61·1	63·5	64·9	67·2	—	—	—
Foot Length	A	18·1	18·8	20·5	21·1	21·6	22·6	23·0	23·6	24·3	25·4	25·5	26·1	26·3	26·1
	C	17·2	17·8	18·9	19·7	20·4	21·4	21·9	22·9	23·7	24·8	25·0	—	—	—
Foot Breadth	A	6·9	7·1	7·6	7·5	7·8	8·0	8·2	8·3	8·6	9·1	9·2	9·5	9·4	9·6
	C	6·8	7·0	7·2	7·4	7·7	8·0	8·2	8·5	8·9	8·8	8·7	—	—	—
Hand Length	A	12·8	13·3	14·2	14·7	15·1	15·6	15·8	16·4	17·0	18·2	18·3	18·9	19·0	19·5
	C	12·4	12·8	13·3	13·7	14·1	14·7	15·2	15·7	16·3	16·3	—	—	—	—
Hand Breadth	A	6·3	6·5	6·8	6·9	7·1	7·3	7·3	7·5	7·8	8·3	8·6	8·9	8·9	8·9
	C	5·9	6·2	6·3	6·6	6·8	7·0	7·2	7·3	7·5	7·9	—	—	—	—
Chest Girth (xiphoid level)	A	55·2	57·0	58·8	59·8	62·0	64·5	—	65·0	69·0	72·6	75·5	78·5	80·4	82·2
	C	54·3	55·4	57·4	59·2	61·0	62·4	63·5	66·2	67·4	—	—	—	—	—
Waist Girth	A	52·1	53·3	55·1	57·8	57·9	59·5	60·0	60·8	62·1	65·5	67·1	68·9	70·3	72·7
	C	52·5	53·0	55·0	55·5	56·8	58·0	59·1	61·0	62·2	64·7	66·1	—	—	—
Hip Girth (iliac level)	A	55·9	57·3	60·3	60·6	61·8	63·3	64·1	66·0	68·3	72·3	73·9	75·2	76·9	79·3
	C	54·7	54·8	57·0	58·8	60·4	62·1	63·6	66·3	66·4	70·7	72·9	—	—	—
Upper Arm Girth	A	15·7	16·1	17·5	18·0	18·4	19·1	19·5	20·0	20·5	22·0	22·7	23·7	23·6	25·1
	C	15·8	15·8	16·5	16·9	17·4	18·0	18·5	19·5	19·6	21·3	21·6	—	—	—
Wrist Girth	A	11·9	11·9	12·7	12·7	12·9	13·2	13·3	13 6	14·3	15·3	15·9	16·4	16·5	17·0
	C	11·8	11·8	12·1	12·3	12·7	13·0	13·3	13·7	13·8	15·1	15·2	—	—	—
Ankle Girth	A	15·2	15·8	16·7	17·4	17·3	18·4	18·6	18·9	19·3	20·7	20·9	21·1	21·2	21·9
	C	14·5	14·7	15·4	15·8	16·3	16·8	17·5	18·1	18·6	20·3	20·2	—	—	—
Knee Girth	A	21·2	21·5	23·5	24·7	25·9	26·5	27·5	28·2	29·5	31·4	31·2	32·0	32·2	32·9
	C	20·5	20·6	21·6	23·0	23·7	24·5	25·9	27·5	28·2	29·0	29·5	—	—	—
Inter-spinous Breadth	A	16·2	17·0	17·4	17·6	18·1	19·3	19·3	19·5	20·2	21·4	21·7	22·1	22·9	23·8
	C	14·9	15·3	15·6	16·4	16·9	17·7	17·9	18·4	18·6	21·4	22·7	—	—	—
Inter-trocanteric Breadth	A	20·1	20·7	22·3	22·7	23·4	24·3	25·0	26·1	27·8	29·5	30·3	31·6	32·4	32·9
	C	19·3	20·0	21·0	21·9	22·7	23·6	24·5	25·4	26·2	28·5	29·5	—	—	—
Inter-acromial Breadth	A	24·7	25·7	27·4	28·0	29·0	29·5	31·0	31·0	32·7	33·9	34·9	36·4	36·8	37·2
	C	23·3	24·0	25·4	26·2	27·3	28·4	29·2	29·8	30·8	33·5	33·8	—	—	—
Number in Group	A	20	26	23	50	82	82	95	86	66	64	70	64	72	23
	C	50	52	55	57	65	60	72	65	81	23	12	—	—	—

APPENDIX TABLE B

MEAN MEASUREMENTS (cm.) GIRLS

Age (yrs.) ..		5	6	7	8	9	10	11	12	13	14	15	16
Weight (kg.) ..	A	19·4	21·6	24·6	28·4	30·4	33·6	39·0	43·8	49·8	55·1	56·7	59·5
	C	18·4	20·5	21·9	23·8	26·4	28·8	31·7	35·6	41·9	45·8	—	—
Standing Height ..	A	113·7	119·4	124·7	131·3	134·2	142·0	147·2	154·5	159·6	163·1	163·3	166·1
	C	107·8	112·2	118·3	122·4	127·5	131·7	137·2	142·8	149·8	152·9	—	—
Sitting Height ..	A	63·8	66·0	68·5	71·1	72·1	75·1	77·5	80·9	84·6	86·6	87·2	88·6
	C	60·5	62·8	65·5	67·3	69·2	71·5	73·7	76·0	79·4	80·9	—	—
Leg Length ..	A	56·3	60·3	63·8	68·5	70·6	75·1	78·3	81·6	84·2	85·1	85·5	87·1
	C	52·7	55·9	59·6	62·4	65·4	67·8	71·5	74·8	78·6	79·8	—	—
Arm Length ..	A	45·4	47·4	50·2	53·3	54·5	57·9	60·5	63·7	66·6	67·7	67·9	69·4
	C	44·2	47·1	49·7	50·8	53·0	54·4	57·1	59·8	63·0	63·4	—	—
Foot Length ..	A	17·8	18·5	19·4	20·6	20·8	21·8	22·7	23·3	23·8	23·9	24·1	24·4
	C	16·8	17·7	18·8	19·4	20·3	20·6	21·6	22·3	23·3	23·4	—	—
Foot Breadth ..	A	6·5	7·0	7·2	7·5	7·7	7·9	8·2	8·3	8·6	8·8	8·7	8·8
	C	6·6	6·9	7·0	7·2	7·5	7·8	8·0	8·3	8·6	8·8	—	—
Hand Length ..	A	12·4	13·2	13·7	14·4	14·7	15·3	16·3	16·6	17·3	17·5	17·5	17·7
	C	12·1	12·7	13·1	13·5	14·0	14·3	14·9	15·7	16·6	16·9	—	—
Hand Breadth ..	A	6·0	6·2	6·4	6·7	6·9	7·1	7·3	7·4	7·7	7·8	7·8	8·0
	C	5·8	6·0	6·1	6·3	6·5	6·7	6·9	7·1	7·5	7·7	—	—
Chest Girth (xiphoid level)	A	53·4	55·4	56·6	59·4	60·2	62·3	65·5	67·2	68·9	71·2	71·7	—
	C	54·3	56·4	56·2	57·8	60·1	61·2	63·0	65·7	66·2	68·0	—	—
Waist Girth ..	A	48·5	50·2	52·0	54·9	55·0	56·3	59·4	60·9	63·5	65·9	66·8	66·9
	C	51·7	53·8	53·3	54·4	56·5	57·0	58·3	60·0	62·3	62·7	—	—
Hip Girth ..	A	58·9	60·8	64·1	68·3	70·5	72·8	77·8	82·2	87·6	92·6	94·9	97·1
	C	58·5	61·7	62·9	65·0	67·8	70·1	72·7	76·5	79·6	85·0	—	—
Upper Arm Girth ..	A	15·7	16·4	17·2	18·1	18·7	19·2	20·5	20·9	22·1	23·0	23·3	24·2
	C	16·0	16·5	16·7	17·2	17·9	18·6	18·9	19·7	20·5	21·2	—	—
Wrist Girth ..	A	11·9	11·9	12·3	12·9	13·0	13·5	14·2	14·5	15·0	15·3	15·5	15·7
	C	11·6	12·0	12·0	12·3	12·5	13·0	13·3	13·5	14·3	14·8	—	—
Ankle Girth ..	A	15·2	15·7	16·5	17·4	17·8	18·1	19·2	19·5	19·6	20·3	20·5	20·8
	C	14·7	15·3	15·6	16·2	16·6	16·9	17·7	18·4	19·2	20·2	—	—
Knee Girth ..	A	21·1	22·0	23·3	24·3	25·2	26·4	28·2	29·4	30·7	32·1	33·0	34·0
	C	20·4	21·5	21·8	22·7	23·9	24·6	25·5	27·0	28·8	30·2	—	—
Inter-spinous Breadth ..	A	15·8	16·5	16·7	17·7	18·0	18·8	19·3	20·5	20·9	21·4	22·0	22·2
	C	14·7	15·3	15·5	16·1	16·8	17·6	17·9	18·6	19·8	21·5	—	—
Inter-trochanteric Breadth ..	A	19·8	20·6	21·7	23·1	23·7	24·7	26·2	28·3	30·2	31·3	31·7	32·6
	C	19·5	20·5	21·1	21·8	22·7	23·5	24·6	26·1	28·0	29·3	—	—
Inter-acromial Breadth ..	A	24·3	25·5	26·6	28·1	28·8	30·1	31·0	32·0	33·5	34·4	34·7	35·1
	C	23·2	25·3	25·3	26·2	26·7	28·1	29·3	30·2	31·2	33·2	—	—
Number in Group	A	12	39	56	53	46	57	53	50	73	71	53	52
	C	61	52	51	70	69	49	63	54	52	55	—	—

A = independent schools. C = council schools (poor districts).

(1) All girths taken by ¼-in. wide steel tape, light pressure (about 2 oz. tension).
(2) Trunk breadths taken with spreading calipers (pelvimeter).
(3) Leg length taken by steel tape from floor to top of great trochanter.
(4) Arm length by steel tape from acromium edge to medius, arm horizontal.
(5) Foot and hand measurements by projection on to squared base.
(6) Knee girth, minimum below tibial tuberosity and head of fibula.

CORRIGENDA

British Journal of Social Medicine (1953), **7**, 162, col. 2:

l. 5 from bottom, *for* Jenkins *read* Jenkin.

l. 2 from bottom, *for* 1935 *read* 1953.

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